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Original Communications

A STUDY OF 244 PREMATURELY BORN INFANTS

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SCOPE AND METHOD OF STUDY

THIS study has been made in an effort to determine the abnormal conditions that occurred in a group of prematurely born infants, especially the conditions that caused death, and to study the possible effects on these infants of complications of their mothers' pregnancy. The study was based on the medical records of all infants prematurely born in the New Haven Hospital during the ten-year period 1924-1933, and included every liveborn infant who was described in the medical records as "premature" and who weighed at birth 2,500 gm. or less. There were 244 such infants.* Sixty-six of them died before discharge from the hospital and 178 were alive at the date of discharge.† Of the 244 infants 112 were males, 132 females; 218 were white, 26 colored. Seventy were patients of private physicians and 174 were ward patients.

One hundred and ninety-four infants were the result of single pregnancies (79 per cent) and 50, of multiple pregnancies (21 per cent). The infants of multiple birth included one set of triplets, 21 pairs of twins, and 5 single twins.‡

Although infants of multiple birth are usually considered in the same category with single born infants with regard to prematurity, provided the birth weight is 2,500 gm. or less, many of them are born close to

*Twelve additional infants were described in the hospital records as premature, but their weights were not known and therefore these infants were not included in the study.

†The information with regard to these 244 infants was obtained only for the period of hospitalization. For the 66 infants who died in the hospital the information covers the entire period from birth to death; for the 178 infants discharged alive, the period from birth to discharge.

From U. S. Department of Labor, Children's Bureau, Washington, D. C., and Department of Pediatrics, Yale University School of Medicine, New Haven, Connecticut.

‡In each of these 5 cases the other twin weighed more than 2,500 gm. and therefore was excluded from the study.

term or even at term and have had a longer period of gestation than single born infants of the same weight, are therefore more mature and so are better prepared for extrauterine life. For this reason, although infants of multiple birth have been considered in this study as part of the *total number* of prematurely born infants, they have been dealt with as a special group also when mortality was studied.

Since it is well known that morbidity and mortality of prematurely born infants are closely related to birth weight, consideration of such infants in groups according to birth weight has become customary. Ylppö,[1] for example, separated the prematurely born infants that he studied into four groups according to weight at birth:

1. Infants weighing from 600 to 1,000 gm.
2. Infants weighing from 1,001 to 1,500 gm.
3. Infants weighing from 1,501 to 2,000 gm.
4. Infants weighing from 2,001 to 2,500 gm.

This grouping has been adopted almost universally in the literature.

In the present study the number of cases is small; therefore the infants have been divided into two large groups according to birth weight, as well as into the four smaller groups according to the plan used by Ylppö. These two large groups, which will be referred to throughout this paper as the "lower weight group" and the "higher weight group," are

1. Infants weighing 1,500 gm. or less (60 infants, 25 per cent of the cases).
2. Infants weighing 1,501 to 2,500 gm. (184 infants, 75 per cent of the cases).

The four smaller groups have been used in this study only for the purpose of studying mortality; the two larger groups, for studying various other factors as well as mortality.

WEIGHT OF INFANTS AT BIRTH

Of the 66 prematurely born infants who died in the hospital 43 were in the lower weight group and 23 in the higher. In Table I the mortality is shown for Ylppö's four weight groups and also for the two groups used throughout this study. The mortality of the infants was found to vary inversely with the birth weight, being 100 per cent among those weighing 1,000 gm. or less; 62 per cent among those weighing 1,001 to 1,500 gm.; 22 per cent among those weighing 1,501 to 2,000 gm., and 6 per cent for those weighing 2,001 to 2,500 gm. When the mortality of the infants was considered according to the two large weight groups used in this study—(1) 1,500 gm. or less and (2) 1,501 gm. to 2,500 gm.—it was found to be more than five times as high among the infants in the lower weight group (72 per cent) as among infants in the higher weight group (13 per cent).

TABLE I

MORTALITY OF PREMATURELY BORN INFANTS BY WEIGHT AT BIRTH

BIRTH WEIGHT	TOTAL	INFANTS WHO DIED		INFANTS WHO LIVED	
		NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
Total	244	66	27.0	178	73.0
1,500 gm. or less	60	43	71.7	17	28.3
1,000 gm. or less	15	15	*	–	–
1,001-1,500 gm.	45	28	*	17	*
1,501 to 2,500 gm.	184	23	12.5	161	87.5
1,501-2,000 gm.	78	17	21.8	61	78.2
2,001-2,500 gm.	106	6	5.7	100	94.3

*Percentage not shown because the number of cases was less than 50.

SINGLE AND MULTIPLE BIRTHS

The mortality for infants who were the result of multiple pregnancies was much lower than for single born infants—16 per cent among the infants born of multiple pregnancies and 30 per cent among the single-born. This difference is similar to that noted by Ylppö,[1] who investigated a much larger group of cases, and probably is due to the greater maturity of the infants of multiple birth.

AGE OF INFANTS AT DEATH

The age at death of the 66 infants who died in the hospital is shown in Table II. Thirty-seven (more than half of them) died in the first twenty-four hours after birth. Eleven infants died in the next four days, 12 in the period between the fifth day and the thirtieth day, and 6 after they were one month old. When the mortality at each of these age periods up to one month was considered in the two weight groups, it was found to be definitely higher in the lower weight group than in the higher, as would be expected.

PRIVATE AND WARD CASES

Among the private cases the mortality for the total group of infants was 30 per cent; among the ward cases, 26 per cent, a difference that is

TABLE II

AGE AT DEATH OF 66 PREMATURELY BORN INFANTS

AGE AT DEATH	WEIGHT AT BIRTH		
	TOTAL	1500 GM. OR LESS	1501 TO 2500 GM.
Total	66	43	23
Less than 24 hours	37	25	12
24 hours, less than 5 days	11	7	4
5 days, less than 1 month	12	8	4
1 month or more*	6	3	3

*Includes one child who died at fifteen months. She was in the hospital during entire period of her life.

not significant statistically. In connection with the similarity in the mortality, it is of importance that in recent years the premature infants born in the private rooms have been transferred for treatment to the special units for premature infants in the pediatric wards and, therefore, both groups receive the same general treatment.

CONDITION OF MOTHERS

The mothers' records* were analyzed to determine:

(1) factors that might have influenced the premature onset of labor, and

(2) factors that might have influenced the fate of the offspring.

Parity.—The parity of the mothers did not seem to affect the weight of the infants or their mortality, for the proportion of infants in the two weight groups was practically the same for the infants of primiparas and of multiparas, and the mortality also was practically the same. The number of primiparas among the mothers was about the same as the number of multiparas.

Complications of Pregnancy.—The possible effects on the infant of the complications of the mother's pregnancy were studied. In 169 of the 244 cases the mothers had had complications of pregnancy; 126 had had one complication only; and 43 had had more than one complication. The cases have been grouped, as shown in Table III, according to the complication which, from the point of view of its severity and the period of pregnancy in which it occurred, was considered the most important in its effect on the infant. For example, a mother who had pyelitis and multiple pregnancy is included in the group of cases with infection.

Toxemia occurred in 73 cases; bleeding in 28; infection in 27; multiple pregnancy alone in 23†; and other complications in 18. A detailed list of the complications, grouped under four main headings, is given in the footnote to Table III.

The mortality of the infants among mothers who had had complications was approximately the same as that among mothers who had had no complication (28 and 24 per cent, respectively). When the infants were considered in the two weight groups, however, it was found that in the higher weight group the proportion of infants who died was about the same when the mothers had had complications as when they had had none (14 and 10 per cent respectively), whereas in the lower weight group the proportion who died was smaller when the mothers had had complications than when they had had none (67 and 86 per cent respectively). The proportion of infants of very low birth weight (1,000

*When prenatal and natal factors were studied, the mother of triplets was counted as three cases; the mother of twins as two cases whenever both twins were included in the study, as one case when only one of the twins was included.

†Twenty-seven additional cases in which multiple pregnancy occurred are included under other groups of complications as in these cases the multiple pregnancy was considered relatively unimportant.

TABLE III

COMPLICATIONS OF PREGNANCY* OF MOTHERS OF PREMATURELY BORN INFANTS BY MORTALITY OF INFANTS IN TWO WEIGHT GROUPS

COMPLICATIONS OF PREGNANCY	BIRTH WEIGHT OF INFANTS								
	TOTAL			1500 GM. OR LESS			1501 TO 2500 GM.		
	TOTAL	INFANTS DIED	INFANTS LIVED	TOTAL	INFANTS DIED	INFANTS LIVED	TOTAL	INFANTS DIED	INFANTS LIVED
Total	244	66	178	60	43	17	184	23	161
No complications	75	18	57	14	12	2	61	6	55
Complications	169	48	121	46	31	15	123	17	106
One complication only	126	36	90	35	24	11	91	12	79
Toxemia	51	11	40	12	6	6	39	5	34
Multiple pregnancy only	23	2	21	2	–	2	21	2	19
Bleeding	22	10	12	10	9	1	12	1	11
Infection	18	4	14	3	1	2	15	3	12
Other	12	9	3	8	8	–	4	1	3
More than one complication	43	12	31	11	7	4	32	5	27
Toxemia and other	22	8	14	8	5	3	14	3	11
Bleeding and other	6	1	5	1	–	1	5	1	4
Infection and other	9	1	8	1	1	–	8	–	8
Other (2 or more)	6	2	4	1	1	–	5	1	4

*The detailed list of complications of pregnancy is as follows:

Toxemia, total		73	
Toxemia alone		51	
Toxemia, type not specified	23		
Nephritic	15		
Preeclamptic	6		
Eclamptic	7		
Toxemia and other complications		22	
Toxemia and multiple pregnancy	9		
Toxemia, multiple pregnancy, and hydramnios	2		
Toxemia, multiple pregnancy, and heart disease	2		
Toxemia and heart disease	1		
Toxemia, heart disease, and ectopic pregnancy	1		
Toxemia and pyelitis	4		
Toxemia and anemia	1		
Toxemia and myomas	1		
Toxemia and bleeding	1		
Bleeding, total		28	
Bleeding alone		22	
Bleeding, type not specified	10		
Placenta previa	4		
Premature separation of placenta	8		
Bleeding and other complications		6	
Bleeding and multiple pregnancy	2		
Bleeding and fall	1		
Bleeding and syphilis	1		
Bleeding and bronchitis	1		
Bleeding and abscess of Bartholin's gland	1		
Infection, total		27	
Infection alone		18	
Gonorrhea	1		
Pyelitis	5		
Chronic cervicitis	2		
Syphilis	4		
Pneumonia	2		
Appendicitis	1		
Rheumatic heart disease	1		
Measles	1		
Tuberculosis	1		
Infection and other complications		9	
Pyelitis and other	4		
Pyelitis and rheumatic heart disease	1		
Pyelitis and chronic arthritis	1		
Pyelitis and multiple pregnancy	2		
Syphilis and other	3		
Syphilis, gonorrheal vaginitis, and myomas	1		
Syphilis and multiple pregnancy	2		
Rheumatic heart disease and other	2		
Rheumatic heart disease and multiple pregnancy	2		
Other complication, total		18	
One complication		12	
Brain tumor	1		
Diabetes	2		
Psychosis	3		
Lacerated cervix	2		
Fall	1		
Anemia	1		
Hydramnios	2		
More than one complication		6	
Hydramnios and multiple pregnancy	5		
Psychosis and multiple pregnancy	1		

gm. or less) was slightly greater among the mothers with no complications. It was found, moreover, that a greater proportion of these mothers had had previous abortions and premature infants. In the total group and in the groups according to birth weight the numbers of infants are small, and the findings are merely suggestive.

The mortality of the infants was not studied according to the different groups of complications because the number of cases in each group was too small. The mortality of the infants, however, appeared to be highest among the mothers with bleeding as a complication (with or without other complications) and lowest among the mothers with multiple pregnancy as the only complication. As would be expected, the largest proportion of infants of low birth weight was found among the mothers in the former group, the smallest among those in the latter.

Complications of Labor and Delivery.—Labor occurred spontaneously in 149 cases and was induced in 61.* When the onset of labor was spontaneous, 29 (19 per cent) of the infants died; when it was induced, 22 (36 per cent) died.

The proportion of infants in the lower weight group was much greater among the mothers whose labor had been induced than among those whose labor had been spontaneous. The proportion of infants in the lower weight group who died was slightly greater also among the mothers whose labor was induced. Labor was generally induced on account of complications of pregnancy.

In 51 cases premature rupture of membranes occurred. In 13 of these cases the infant died. In only 7 of these 13 cases was the death of the infant ascribed to infection (general septicemia, 2 cases; respiratory infection, 3; diarrhea, 2). Only one of the 38 infants who lived had an infection (gonorrheal ophthalmia).

The total duration of labor† (first and second stages combined) was known in 190 cases. *Precipitate or prolonged labor* occurred in 42 cases (22 per cent); the labor was regarded as of normal duration in 148 cases (78 per cent). Among the 190 cases primiparas and multiparas were about equally represented (93 of the former, 97 of the latter). The frequency of normal labor was practically the same among primiparous and multiparous women (51 and 49 per cent, respectively). The proportion of infants who died was about the same among the group of mothers in whom labor was normal in duration as among those in whom it was abnormal (20 and 21 per cent, respectively).

Among the mothers whose labor was of normal duration, the deaths of their infants occurred in about the same proportion in the primiparas

*In 15 cases there was no labor (cesarean section); in 19 cases the type of labor was not recorded.

†Labor was regarded as normal if its duration was more than three but less than thirty hours; abnormal, if three hours or less, or thirty hours or more. The number of cases was insufficient and the data inadequate to permit study of the first and second stages of labor separately.

and in the multiparas (21 and 18 per cent, respectively). Among the mothers whose labor was of abnormal duration, however, the deaths of their infants occurred about three times more frequently in the primiparas than in the multiparas (35 and 12 per cent, respectively). Twenty per cent of the infants born after a normal duration of labor were in the lower weight group, 80 per cent in the higher. These proportions were practically the same for infants born after an abnormal duration of labor.

When the duration of labor was normal, nearly two-thirds of the infants in the lower weight group died (19 of 30 infants); in the higher weight group a very small proportion of the infants died (10 of 118 infants). On the other hand, when the duration of labor was abnormal, a larger proportion of infants in the lower birth weight group died (6 of 8 infants), but the proportion of deaths among infants in the higher weight group was the same (3 of 34 infants).

Abnormal pelvis was found in 19 cases, recorded as "contracted" in 9 cases, "rachitic" in 3, "funnel" in 5, and "simple flat" in 2. In 3 of the 19 cases the infant died; 2 of these infants were in the lower weight group and 1 in the higher. Sixteen of the infants lived; 1 was in the lower weight group and 15 in the higher. In none of the 19 infants were evidences of birth injury found.

Type of Delivery.—There were 165 cases of *spontaneous delivery* and 79 of *operative delivery,* as shown in Table IV. Of the infants born spontaneously, 45 (27 per cent) died; of those born by operative procedure, 21 (27 per cent) died. Thus mortality among these premature infants did not appear to depend upon whether delivery was operative or spontaneous. Of the infants born spontaneously, more than one-fourth were in the lower weight group; of the infants born by operative procedure, one-fifth. The number of cases was too small to permit statistical study of the mortality of the infants in relation to the different types of operative delivery. The largest proportion of infant deaths, however, occurred when the mother had been delivered by cesarean section.

TABLE IV

TYPE OF DELIVERY OF MOTHERS OF PREMATURELY BORN INFANTS BY MORTALITY OF INFANTS IN TWO WEIGHT GROUPS

TYPE OF DELIVERY	TOTAL	BIRTH WEIGHT OF INFANTS					
		1500 GM. OR LESS			1501 TO 2500 GM.		
		TOTAL	INFANTS DIED	INFANTS LIVED	TOTAL	INFANTS DIED	INFANTS LIVED
Total	244	60	43	17	184	23	161
Spontaneous delivery	165	44	29	15	121	16	105
Nonspontaneous delivery	79	16	14	2	63	7	56
Forceps	25	1	1	-	24	1	23
Cesarean section	20	5	5	-	15	3	12
Version and extraction	17	5	4	1	12	2	10
Breech extraction	17	5	4	1	12	1	11

CAUSES OF ILLNESS AND OF DEATH OF INFANTS

In Table V are shown the principal causes of death of the 66 infants who died, divided into the two weight groups.

TABLE V

PRINCIPAL CAUSE OF DEATH OF 66 PREMATURELY BORN INFANTS

CAUSE OF DEATH	WEIGHT OF INFANT		
	TOTAL	1500 GM. OR LESS	1501 TO 2500 GM.
Total	66	43	23
Prematurity alone	34	27	7
Infection	16	8	8
Intracranial hemorrhage	11	6	5
Congenital defect	3	1	2
Other	2	1	1

Premature birth was considered the principal cause of death* of 34 infants (27 of these were in the lower weight group and only 7 in the higher). Four of these 34 infants were the result of multiple pregnancies (3 in the lower weight group and 1 in the higher). In 14 of the 34 cases postmortem examinations had been made and in each case no cause of death other than the incomplete development of the infant was reported.

Infection was considered the principal cause of death in 16 infants (pneumonia, 6 cases; septicemia, 5; respiratory infection, 2; diarrhea, 2; meningitis, 1); 8 of these infants were in the lower weight group and 8 in the higher. Three of the 16 infants were the result of multiple pregnancies (1 triplet and 2 twins); 1 was in the lower weight group and 2 in the higher. Postmortem examinations were made in 11 cases, and evidence of infection was found in 9 of them.

Intracranial hemorrhage, the result of birth injury, was considered the principal cause of death in 11 infants. Six of these were in the lower weight group, 5 in the higher; 1 of the infants in the higher weight group was a twin. Postmortem examinations confirmed the clinical diagnosis in every case. The deliveries were spontaneous in 10 cases, operative in 1.

Congenital defects were considered the principal cause of death of only 3 infants. One infant, weighing 960 gm. at birth, was an anencephalic monster; 1, weighing 1,625 gm., had multiple malformations of the skeletal and muscular systems; 1, weighing 1,790 gm., had a defect of the abdominal wall with herniation of the viscera. All these infants were single born. Postmortem examinations were not made in any of these cases.

Other conditions not classified in the groups mentioned in the preceding paragraphs caused the death of 2 infants, both of whom were single born. One of them, an infant weighing 1,960 gm. at birth, died of

*Diagnosis based on clinical or pathologic examinations or both.

peritonitis following rupture of a gastric ulcer; the other, weighing 1,282 gm. at birth, died of hemorrhagic disease. Postmortem examinations confirmed these diagnoses.

In no case was the principal cause of death *atelectasis*. Thirty of the 66 infants who died had atelectasis, diagnosed in 22 infants by clinical examination; in 8 of these the diagnosis was confirmed by postmortem examination. Atelectasis was found at postmortem examination in 8 additional infants in whom it was not found clinically. Among these 30 infants more than three times as many were in the lower weight group as in the higher. There were no multiple births in this group of infants.

An effort was made to find out the effect on the onset of respiration in the infant when *morphine* was given to the mother. In 188 cases the history showed whether or not the mother had received morphine after admission to the hospital. Of the 63 infants whose mothers had received one or more doses of morphine, 38 per cent died; and of the 125 infants whose mothers had received no morphine, only 23 per cent died, in spite of the fact that among the infants who died the proportion of infants of low birth weight was slightly greater among the infants of mothers who had had no morphine. When the mother had received morphine, the onset of respiration in the infant was delayed in 41 per cent of the cases; when the mother had not received morphine, in only 16 per cent.

Among the 178 surviving infants, there were 100 who during the period in the hospital had no abnormal conditions nor illnesses (94 of these were in the higher weight group, 6 in the lower) and 78 in whom abnormal conditions were found—101 conditions altogether. Those found most frequently were infection, 32 times; atelectasis, 23 times; congenital defects, 18 times; birth injuries, 5 times; hemorrhages other than intracranial, 2 times; miscellaneous conditions, 21 times.

The surviving infants were discharged from the hospital after various periods of time, whenever the medical staff considered that they might be treated as normal full-term infants. The period in the hospital varied considerably, the average being forty-three days. There were no surviving infants in the lower weight group who remained in the hospital less than eight weeks, but 93 per cent of those in the higher weight group were discharged in less than eight weeks.

Sixty-two per cent of the infants weighed 2,500 gm. or more when discharged; 14 per cent of these had been in the lower weight group at birth and 86 per cent in the higher.

Although these infants may have made return visits to the pediatric clinic following their discharge from the hospital, the records of these visits have not been analyzed for this study nor have records of mental tests been obtained.

METHODS OF CARING FOR INFANTS

The methods used in the care and treatment of the prematurely born infants included in the study varied widely in the ten-year period cov-

ered. The methods have been evolved gradually, and there is no sharp line of demarcation between the periods when different methods of treatment were used. In the early years very few prematurely born infants were transferred from the private pavilion to the pediatric service; now nearly all such infants are so transferred. Air-conditioned rooms were installed in 1928, and other details of treatment have been changed from time to time. For these reasons, it is not possible to compare the results of one method of care and treatment with another in this series of cases. The mortality for the first five-year period was the same as that for the second (27 per cent). Moreover, when the mortality was studied in the two periods for each of the two weight groups, no significant difference was found.

The method used in the most recent period for caring for prematurely born infants in the New Haven Hospital is as follows: A house officer who is a member of the pediatric staff is present at all premature deliveries. The infant is placed immediately after birth in a Hess bed heated to 80° F., which is always kept in readiness in the delivery room. When the house officer considers that the infant's condition will permit a change, the infant is transferred, usually in a Hess bed, to one of the special rooms of the pediatric ward. These rooms, about 10 by 14 feet in size, are air-conditioned, the temperature being automatically maintained by the Yaglou apparatus at about 75° F., and the moisture at about 68 per cent. Efforts are made to maintain the infant's temperature at 37° C., usually by regulation of the temperature of the Hess bed. Nurses and doctors are the only persons admitted to the room; isolation technic is used; hands are washed, and gowns are worn. Nursing care is given by student nurses under the supervision of the head nurse of the pediatric service. The infants are handled as little as possible. Oil baths are given every other day.

The infant is fed in the Hess bed (with the head end of the mattress raised) by means of a medicine dropper with a soft rubber catheter tip or a bottle and nipple of small size. Gavage is resorted to only in very exceptional cases. The feedings consist of breast milk, if it is available, otherwise of modified cow's milk (skimmed or half skimmed) with 5 to 10 per cent dextrimaltose added. The feeding interval is usually two hours. The general principle is to give small feedings at first, and to increase the feedings gradually both in amount and caloric value to an upper limit of about 150 calories daily per kilogram of body weight. Water is given with a medicine dropper between feedings, the total fluid intake being estimated on the basis of about 150 c.c. daily per kilogram of body weight. If the infant cannot take this amount of fluid by mouth, saline solution is administered by hypodermoclysis. Transfusions of blood are given repeatedly if the infant is not doing well or if evidences of infection or other pathologic conditions appear.

Viosterol, 10 to 15 drops daily, and orange juice, 8 c.c. daily, are started near the end of the second week; cod liver oil, 4 c.c. daily, at one to two months of age.

Anti-anemic therapy is not given as a matter of routine.

SUMMARY

This study was based on the medical records of all infants born prematurely in the New Haven Hospital during a ten-year period, and included every liveborn infant who was described in the medical records as "premature" and who weighed at birth 2,500 gm. or less. There were 244 such infants, of whom 66 died without having been discharged from the hospital and 178 were discharged alive. More than half the deaths occurred within twenty-four hours of birth.

The mortality was five times as high among the infants weighing 1,500 gm. or less as among those weighing 1,501 to 2,500 gm. It was nearly twice as high among single born infants as among those the result of multiple pregnancy.

The mortality among the infants of the mothers who had had one or more complications of pregnancy was not significantly higher than among the infants whose mothers had not had complications.

The infant mortality did not seem to be influenced by the duration of labor—normal or abnormal; nor by the type of delivery—spontaneous or operative; but it was higher when labor had been induced than when it had occurred spontaneously.

More than half the infant deaths were reported as caused by prematurity, about one-fourth by infection, and about one-sixth by intracranial hemorrhage.

Atelectasis occurred in nearly half of the infants who died and in about one-seventh of those who survived.

The effect on the fetus of morphine given to the mother was shown by the fact that of the 63 infants whose mothers had received morphine, 38 per cent died; and of the 125 whose mothers had not received morphine, only 23 per cent died, in spite of the fact that among the infants who died the proportion of infants of low birth weight was slightly greater among the infants of mothers who had had no morphine. When the mother had received morphine, the onset of respiration in the infant was delayed in 41 per cent of the cases; when the mother had not received morphine, in only 16 per cent.

In studying the effects on the infant of complications of the mother's pregnancy, labor, and delivery and the results of various abnormal conditions in the infants, the birth weights of the infants should always be taken into consideration and the proportion of those of higher or lower weight known.

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THE LIPOID FACTOR IN GLYCOGEN STORAGE DISEASE

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ALTHOUGH quite a number of clinical reports dealing with glycogen storage disease have appeared in recent years, there are relatively few anatomic observations. In the latter, concomitant fatty disturbances have not been emphasized. In a disease characterized by abnormal storage of glycogen with impaired glycogenolysis, manifesting itself by a very low fasting blood sugar, with practically no response to adrenalin, but with marked sensitivity to insulin, it might be expected that fat metabolism would also be affected. Particularly would this be true in the fasting state or when metabolic activity was increased. That this is so is evident from observations obtained from convincing clinical cases, as well as from those in which the diagnosis was established by biopsy or by necropsy. Acidosis and acetonuria are frequent, especially in the fasting condition. Likewise, elevated values for the blood lipoids have frequently been reported. It is also known that, following the ingestion of a fatty test meal, there is a prolonged, elevated blood lipoid curve. This has been emphasized in distinguishing this disease from other forms of hepatomegaly, particularly in cirrhosis of the liver (Beumer and Loeschke,[1] Unshelm[2]).

At present, there is reason to believe that this disease may occur without hepatonephromegaly as originally described by von Gierke,[3] hence the use of the broader term "glycogen storage disease." There have been types with greatly enlarged hearts (formerly included with idiopathic hypertrophy of the heart in infancy) due to massive deposits of glycogen. In most instances these have been associated with simultaneous abnormal storage in other organs, unaccompanied, however, by any degree of enlargement. Whether in these, biochemical changes similar to those of the hepatomegalies will be found remains as yet to be determined. They have only been recognized at autopsy without previous complete studies.

In the first of von Gierke's two cases of hepatonephromegaly (aged eight years) in addition to massive glycogenic infiltration, there was a considerable amount of fat in the liver. This was present in the form of smaller and larger droplets, infrequently larger than half the cell and often absent in many cells, particularly periportally. These droplets stained positively with sudan, were red with nile blue sulphate and did not stain with Smith-Dietrich or Ciaccio stain. In his second

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case (aged four years) there were larger fatty droplets in the pericentral cells but in all less stainable fat than in the first. In Loeschke's[4] living case of hepatomegaly (aged three years) a biopsy of the liver was obtained. Putschar's description of this deals solely with the deposition of glycogen, intimating that there could not be much fat in view of the amount of the former substance. Schmincke examined a biopsy of the liver in one of Schall's cases[5] (aged eight and one-half years). Fat is not mentioned in the description although there was considerable glycogen in the cells and nuclei. Putschar[6] reported a case (aged four months) with marked glycogenic infiltration of the heart (greatly enlarged) associated with mild deposition in the slightly enlarged liver and moderate in the kidneys. There was no demonstrable fat in the heart or the kidneys, but there was some in the liver, with more or less uniformly distributed medium sized droplets. In Kimmelstiel's case[7] (aged one and two-thirds years) there was abnormal glycogen storage in the liver, brain, striated and cardiac muscle, and to a lesser degree in the kidneys. There was a moderate amount of fat in the liver in the form of large droplets. In Pompe's case[8] of cardiac hypertrophy (aged seven months), there was less stainable glycogen in liver and kidneys. Fatty changes are not mentioned except their absence in the heart. Antopol, Heilbrunn, and Tuchman[9] described a case (aged four and one-half months) similar in many ways to that of Pompe's. There were large fat-containing vacuolated cells in the periportal areas of the liver. There was no fat in the hypertrophied cardiac musculature except for some in the interstitial tissues. There was little fat in the livers of 3 cases (four, five, and eight months, respectively) reported by Humphreys and Kato[10] in which cardiac hypertrophy due to glycogen storage was the main finding. Lastly in Faber's case[11] of hepatonephromegaly (aged one month) there were occasional fat vacuoles, not exceeding the size of the nucleus in the pericentral cells of the liver. There were medium sized fatty droplets in the cells of the convoluted tubules and Henle's limbs of the kidney, always situated at the basal end of the cell, between the nucleus and the basement membrane.

Except in Humphreys and Kato's third case, in which large pale cells were observed in the corpuscles and pulp of the spleen, there is no reference to similar findings in that organ or elsewhere by any of the others.

Although it is realized that the liver ordinarily contains a variable amount of fat, its wide distribution in a greatly enlarged organ would exceed the usual quantitative variables. In two of the instances to be reported here, there were also apparently fat-laden monocytes in spleen and bone marrow, and in one, to a degree comparable with that encountered in diabetic lipemias.

CASE 1.—B. S., aged five months, was born after a difficult instrumental delivery. He fared poorly the first few days but rapidly recovered thereafter. The family history was irrelevant. There was another sibling, ten years of age, who was apparently normal. The parents had noted that the patient's abdomen had been large since birth and had progressively increased in size. Two months prior to the present admission he had a cough and nasal catarrh from which he recovered after two weeks of treatment. He had another upper respiratory infection a week before admission. Physically the child was well developed and nourished. His breathing was labored and rapid with an occasional cough. He was perspiring profusely. There were diffuse coarse moist râles throughout both lung fields with harsh breath sounds everywhere. The heart was not enlarged. The sounds were regular, rapid, and of good quality. The abdomen was greatly enlarged. The liver occupied practically the whole right side to within one inch of the pelvic brim and extended over to the left for 5.5 cm. below the costal margin. The spleen was questionably palpable. There was a diffuse fine maculopapular skin rash. There were palpable small cervical glands bilaterally.

The temperature rose from 100° F. on the day of admission to 108° F. terminally on the third day. During this time, he showed signs of marked acidosis, with considerable respiratory embarrassment. There was a note on the third day that blood withdrawn for chemical analysis was creamy and opaque grayish yellow in color.

There were the following additional findings: body weight, 12.3 pounds; height, 25½ inches; circumference of the head, 17¼ inches; circumference of the chest, 17 inches; and circumference of the abdomen, 19½ inches; blood pressure, 132/60; red blood cells, 3,510,000 with 63 per cent hemoglobin; white blood cells, 17,300 to 24,900 with 50 per cent polymorphs, 42 per cent lymphocytes, 7 per cent monocytes, 1 per cent myelocytes; fasting blood sugar, 51 mg. per cent; carbon dioxide, 24 volumes per cent; nonprotein nitrogen, 48 mg. per cent; serum protein, 8.4 gm. per cent; chlorides, 102 c.c.N/10, Cl/100 c.c.; cholesterol, 394 mg. per cent; calcium, 13 mg. per cent; ionized calcium, 5.3 mg. per cent; phosphorus, 7 mg. per cent; sodium, 136 milliequivalents / liter. The urine contained acetone with a very slight trace of albumin. There was no sugar. *X*-ray films of the extremities revealed transverse bands 2 to 3 mm. from the ends of the long bones and of the chest accentuated peribronchial markings.

Necropsy was performed ten hours postmortem. There was a slight amount of pitting edema over the lower legs and ankles. The subcutaneous fat was pale yellow in color and approximately 4 mm. in thickness. There was no free fluid in the peritoneal cavity. The serosal surfaces were smooth and glistening. The liver was markedly enlarged (Fig. 1), extending far over to the left and lying within the iliac fossa in the right axillary line. The stomach and intestines were pushed inferiorly and to the left. The spleen was not enlarged and was well above the costal margin. The kidneys, as seen in situ, appeared large. Pleural and pericardial cavities were not remarkable.

**Heart* weighed 35 gm. (29). There was very little epicardial fat. The myocardium of the left ventricle was somewhat thickened, measuring on the average 0.9 cm. (average normal at this age, 0.7). It was pale reddish brown in color. Except for dilatation of the tricuspid ring (5 cm. as compared with average normal of 4 cm.), the heart was otherwise not very remarkable.

Lungs.—The right lung weighed 64 gm. (38); the left, 42 (35). The dependent and posteroinferior portions of both lungs were deep dark purplish red in color, and only slightly aerated. The cut surfaces were moist and from them a small amount of serosanguineous fluid could be scraped. The anterior portions of the lungs were

*Here and in the remainder of this paper the figures in parentheses will represent average weights for the organs as given in the tables of Coppoletta and Wolbach (Am. J. Path. 9: 55, 1933).

pink, dry and air containing. The bronchi contained a small amount of serosanguineous frothy fluid. Their mucosal surfaces were pale yellowish white in color.

Spleen weighed 15 gm. (16). It was firm in consistency with a thin transparent finely wrinkled capsule. The cut surfaces were pale yellowish red with numerous coarse purplish markings.

Liver weighed 705 gm. (188). It was firm with a smooth, thin and transparent capsule. Both the external and cut surfaces were strikingly pale yellowish in appearance, resembling that of a marked fatty liver, with these differences, however: the surfaces were wet; from them a grayish fluid could be scraped; and the liver substance was less friable. The usual markings were in great part obscured. The

Fig. 1.—The greatly enlarged liver occupying both upper quadrants and lying in the iliac fossa on the right.

gallbladder was small and contained approximately 1.5 c.c. of golden brown bile. Its wall was thin and the mucosa, smooth and velvety. The biliary ducts were not remarkable.

Kidneys.—The right kidney weighed 46 gm. (25), the left, 48 gm. (25). They were firm, surrounded by a moderate amount of fat, and covered by thin capsules which could readily be stripped. Both the smooth external, as well as the cut, surfaces were strikingly pale in appearance. The usual markings were preserved.

Brain weighed 655 gm. (422). Except for mild convolutional flattening and a moderate pressure cone, the brain, meninges and calvarium were not remarkable.

Gastrointestinal Tract.—Pancreas weighed 8 gm.; adrenals (combined weight), 5 gm.; thyroid, 2 gm.; thymus, 12 gm.; pituitary, pineal, middle ears, air sinuses, bones, and aorta were grossly not remarkable.

Portions of liver, kidney, heart, spleen, pancreas, adrenal, skeletal muscle, brain, and lung were fixed in absolute alcohol. These were stained by Best's stain for glycogen. Representative samples of the above and other organs were fixed in formalin and Zenker's solution and subsequently stained with hematoxylin eosin. Various other stains were employed, using when necessary the material fixed in the three solutions and applying them to the organs indicated in the histologic description: for connective tissue, Mallory's aniline blue and phosphotungstic acid hematoxylin; for fat, scharlach R, nile blue sulphate, Smith-Dietrich, osmic acid, Fischler, Schultz, and Ciaccio; for glycogen, Langhan's iodine; for calcium, von Kossa; for iron, Tirmann's Turnbull blue, Mallory's ammonium sulphide, and potassium ferricyanide, Hueck's modification of Turnbull's blue; for bacteria, Gram and Giemsa; Foot's stain for reticulum; and a modified aniline blue, to bring out the granular qualities of the isletal cells of the pancreas.

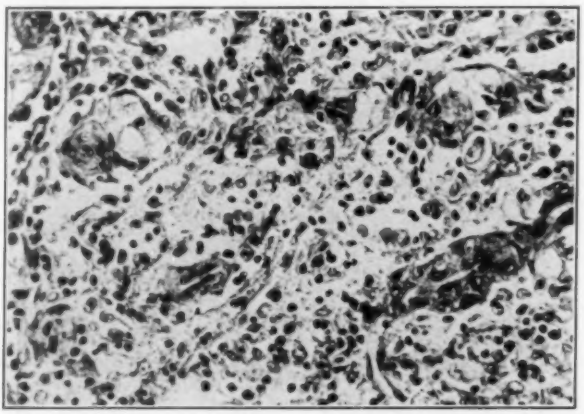

Fig. 2.—Spleen (hematoxylin and eosin). Note the large pale cells surrounding and compressing the arterioles. These were histochemically inert, staining neither for glycogen nor for fat. (×1250.)

Microscopic Examination

Heart.—The muscular fibers were of fairly uniform thickness with, in many, clear, large perinuclear spaces. These in instances contained pigmented granules. Only occasional fibers, but many leucocytes, contained glycogen. There was no iron or fat.

Lung.—There was only one lobular area of pneumonia with seropurulent exudate. Here were few scattered intracellular bacterial rods and cocci. Elsewhere there was moderate atelectasis with dilated bronchioles and areas of emphysema. There were scattered areas of alveolar hemorrhage and in others monocytes and occasional polymorphonuclears. The alveolar septal capillaries were congested and tortuous with increased numbers of polymorphonuclears and monocytes. There was no glycogen, except in an occasional polymorphonuclear. The free alveolar monocytes contained fine fatty droplets and only rarely hemosiderin granules.

Spleen (Fig. 2.).—There were many large pale cells, intimately associated with the intralobular arterioles, occurring singly and far more frequently in groups, within the pulp cords as well. These cells varied from 13.5 to 19.7 $\mu\mu$ in diameter. They were spherical or oval in shape with fairly sharply defined borders. The cytoplasm was frequently finely honeycombed with, in instances, larger vacuolated spaces. At times the cytoplasm appeared clear with a few strands attached to the nucleus.

The latter was usually single, rarely double, eccentrically placed in the cell, oval or spherical in shape, although occasionally wrinkled or slightly lobated and contained fine chromatin threads. These cells were incorporated in the parietes of intralobular arterioles, often disorganizing the muscular fibers and lying in juxtaposition to the endothelium, or compressing the muscular coat and thereby enlarging the adventitial portion of the vessel. In almost all instances the lumen was

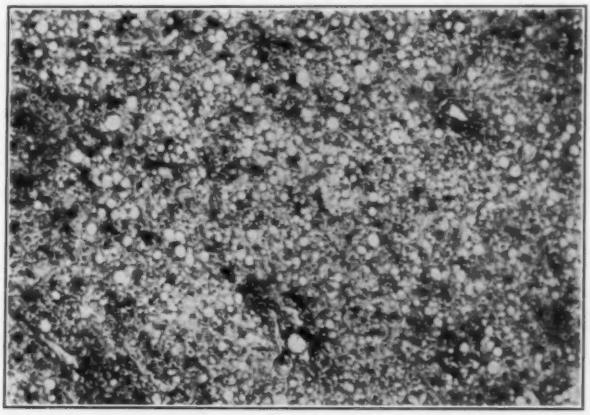

Fig. 3.—Liver (hematoxylin and eosin). An average field. Note the sharply bordered polygonal cells and the many large vacuolated ones. There was marked infiltration of the cells with glycogen and fat. (×150.)

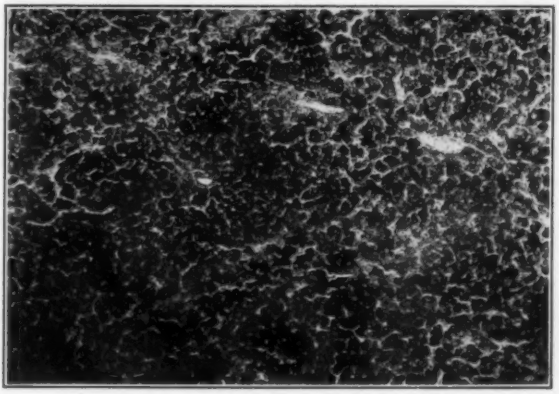

Fig. 4.—Liver (scharlach R). Note the abundance of fat. (×80.)

compressed or obliterated. Where related to these vessels, these cells were surrounded by fine collagenous fibrils. In all instances they were histochemically inert. Neither stains for glycogen nor repeated and varied stains for fat were to the slightest degree positive. They were not birefringent. There was very little blood in sinusoids or pulp cords. The former, however, were patent with prominent, almost columnar, lining endothelial cells. The subendothelial reticulum was prominent. There were large numbers of hemosiderin-laden monocytes, concentrated chiefly about trabeculae and intralobular vessels, so that with iron stains their dis-

tribution almost defined irregular lobelike areas. Here and there were increased numbers of polymorphonuclears. The corpuscles were of medium size with fairly large reactive central foci, made up of phagocytic reticular cells with included and scattered nuclear débris. These cells frequently contained sudanophilic granules.

Pancreas.—The islets were abundant and generally large. The cells were in general larger than in a control series of the same age. There were not infrequent cells with very large giant nuclei, from two to three times normal size, which were rich in chromatin. There was also moderate variation in nuclear size of the other cells. With modified aniline blue most of the cells stained light red, some deeper red, and fewer deep blue. The proportions of these were about the same in the control group. The cells with the larger nuclei, for the most part, stained blue. The parenchymal elements were not remarkable except for rare intracytoplasmic inclusion bodies. In some blood vessels and some polymorphonuclears there were deposits of glycogen. Occasional monocytes in the interlobular tissues were strongly positive for iron.

Liver (Figs. 3 and 4).—The lobular outlines were rather poorly defined. The central veins were frequently not seen or were irregularly related to the lobules. There was no increase in portal connective tissue, but there were collagenous fibrils radiating away from these compressed and elongated portal zones. Similar stellate projections of collagen extended from the adventitia of the hepatic veins. The reticular framework was not altered to any degree. The portal and hepatic veins were moderately congested. The sinusoids were narrowed and compressed. The bile ducts and capillaries were here and there dilated but were free of inspissated contents. The liver cells were generally large, plantlike in type, with thick-rimmed, sharply defined borders. They were polyhedral in shape and occasionally so arranged as to form pseudoacinar or tubular structures. There were frequent groups of larger vacuolated cells. The others had a fine threadlike and granular cytoplasm with variable numbers of smaller and larger vacuolated spaces. There were numerous fine granules of glycogen throughout the cytoplasm, occasionally larger in size or in laked form. In the large vacuolated cells, the deposits of glycogen either skirted the periphery or lay within a peripheral rim of cytoplasm surrounding an unflattened nucleus. Occasionally they formed a distinct film about the vacuole. There was massive fatty infiltration in many sections taken from different parts of the organ. Here and there periportal or perivenous (hepatic) areas were spared. The fat was deposited both small and medium sized droplets, and in the large cells as a single conglomerate sphere. It stained deep red with scharlach, rose violet to violet blue with nile blue sulphate, and not at all with the other stains. The nuclei of the cells were moderately large, spherical and more frequently eccentrically placed. Most of them were fairly densely chromatic. There were many, however, that were large and vacuolated, particularly periportally. These often contained glycogen in laked or in spherical form. Occasionally the liver cells were binucleated and, at times, one nucleus was large and vacuolated, the other of normal size and appearance. There was diffusion of glycogen into vascular spaces. There were rare small foci of necrosis with polymorphonuclear infiltration. These cells, as well as polymorphonuclears elsewhere, contained glycogen. The Kupffer cells were not particularly active, often were positive for iron, and occasionally contained sudanophilic granules.

Kidney (Fig. 5).—The cells of the convoluted tubules were of plantlike type. They were of increased height with thick, sharply defined cytoplasmic membranes, appearing in all quite rigid, although occasionally indented. Their central ends were rounded. They appeared hollow (hematoxylin and eosin) with, however, transitional forms, where the vacuolation appeared to start toward the central end of the cell, gradually effacing the acidophilic granular cytoplasm supra- and subsequently subnuclearly. The cells of the distal convoluted tubules were in all more

involved than those of the proximal. The nuclei were basally placed, not flattened but spherical, with fairly thick membranes and compact chromatin. They were rarely enlarged and vacuolated. All these cells were stuffed with glycogen in the form of small and medium sized granules. Extending from the cortex well into the medulla, there was considerable glycogen in the lumina and cells of the limbs of Henle. There were deposits of urates in some of the terminal collecting tubules and hyaline casts, staining positively for calcium within small atrophic tubules, adjoining atrophic glomeruli. In the medulla there were occasional fibrosed areas, distorting adjoining collecting tubules and containing amorphous débris. There were fairly frequent fatty droplets within the convoluted tubular epithelium and occasionally within a glomerular tuft as a spherical mass probably occupying the lumen of a capillary. Many of the glomeruli were of fetal form, the others were not remarkable. Some of the pelvic epithelial cells contained glycogen.

Adrenals.—The adult cortex was regular and moderately thick. There was a greater amount of fat in the glomerular than in the fasiculate zone, but in all a goodly degree of lipoid depletion. The fetal cortex was almost completely involuted,

Fig. 5.—Kidney (hematoxylin and eosin). Note the thick, sharp bordered, rigid appearing, and clear cells of the convoluted tubules. These were massively infiltrated with glycogen. (×170.)

represented only by a highly vascularized fibrillar stroma in which there were many histiocytes laden with fat and hemosiderin.

Lymphoid Tissues.—There were large reactive foci in the primary follicles of the tonsils, paratracheal, mesenteric and peripancreatic lymph nodes, resembling those of the splenic corpuscles. In the tonsil there was a hyaline thrombus in one subepithelial venule and recent mural thrombi in some of the deeper-seated blood vessels. In one small paratracheal lymph node, there were many monocytes, some with a finely honeycombed cytoplasm resembling the pale cells in the spleen. In all nodes there were variable numbers of actively phagocytic monocytes. Some of these and the reticular cells of the reactive foci contained sudanophilic granules.

Thymus.—There was practically no involution. There were relatively few phagocytic monocytes. Within these, Hassall's corpuscles, and in many blood vessels there were deposits of fat. Eosinophilic myelocytes were frequent within the interlobular stroma and scattered throughout the lobules.

Thyroid.—The acini were smallish with little pale staining, at times vacuolated, colloid. The epithelium varied in height, being rather tall, with pale, almost vacu-

olated, cytoplasm at times, and with moderately chromatic nuclei occupying the greater part of the cell. There was no demonstrable fat.

Pituitary.—It was essentially normal, with abundant, acidophilic and chromophobe cells, but fewer basophiles (modified aniline blue).

Adipose Tissues.—In general there was mild to moderate atrophy of the retroperitoneal and mesenteric fat, with smaller lobules and cells.

Osseous System.—Sections of rib and vertebrae were studied. In the former there was a series of transverse bony bars (arrest of growth), one next to the epiphyseal zone. At this zone there was fairly active and regular penetration of the cartilaginous columns by vascularized marrow with, however, slight retardation in osteoid deposition. The nuclei of the cartilage cells in the hypertrophic zone were in instances prominently vacuolated and enlarged. There was some evidence of irregular absorption of the bony trabeculae of the shaft. Periosteum and cortical bone were not remarkable. In the marrow of both ribs and vertebrae there were many large pale cells, identical with those of the spleen. In addition there were occasional actively phagocytic histiocytes. The marrow elements were abundant, chiefly granulocytic, with many lymphocytoid cells, presumably red blood cell precursors. There were relatively few mature myeloid or erythroid forms. There were many megakaryocytes.

Brain and Cord.—The meninges were congested and lightly infiltrated with lymphocytes, monocytes, and an occasional polymorphonuclear. Except for a small area of red blood cell extravasation in the medulla, with mild congestion and edema in all sections, there were no unusual features. There was no demonstrable glycogen.

Pineal.—It appeared to be consistent in development with the age of the child.

Testes and aorta were not remarkable. There were stainable granules of glycogen in some of the striated fibers of the tongue and skeletal muscle. There was a mild degree of inflammatory reaction involving the mucosa of the trachea.

Anatomical Diagnosis.—Glycogen infiltration of liver and kidneys, marked; fatty infiltration of the liver; lobular pneumonia, mild; probably lipoid-storing cells in spleen and bone marrow; mild acute tracheitis; focal liver necroses; toxic reactive changes in lymphoid tissues generally; hemosiderosis of spleen, marked; mild peripheral and cerebral edema; lipoid depletion of adrenal.

CASE 2.—R. R., aged one year (Jewish), was admitted to the hospital for investigation of a large abdominal mass. At the age of two months, a midline dermoid tumor of the scalp was removed. Again at six months, he was seen in this hospital for hoarseness. On both these occasions there were no unusual physical findings. He had not been seen again until two days prior to the present admission. For the preceding six months, however, he had been almost constantly hoarse and had had frequent colds. The pertinent physical and laboratory findings were: rapid respirations; temperature, 100.2° F.; protuberant abdomen with a regular mass, presumably liver, filling the right flank and upper quadrant, extending to within 1 inch of the umbilicus and well over to the left; light trace of albumin in the urine with 3+ acetone (which was always present in subsequent specimens); moderate anemia. On the second day after admission, he was markedly hyperpneic and vomited several times. He died on the third day.

Necropsy was performed one hour postmortem. The incision was restricted to the abdomen. The body was well developed and nourished, measuring 73 cm. in length (normal). The head measured 17 inches in circumference; the chest, 17.5 inches; the abdomen, 20 inches. There was no free fluid in the peritoneal cavity. The liver was greatly enlarged, extending below the level of the crest of the ileum on the right to within 3 cm. of the umbilicus in the midline and disappearing behind the costal margin in the left midclavicular line. The spleen was not enlarged. Both kidneys, however, were greatly increased in size. The pleural and pericardial cavities were not remarkable.

Lungs.—The right lung weighed 104 gm. (64); the left, 74 gm. (57). Both lungs were firm and nodular in the dependent and juxtahilar regions, with dry purplish gray cut surfaces. The larger bronchi were not remarkable. From the smaller branches a serohemorrhagic fluid escaped.

Spleen weighed 32 gm. (26). The capsule was thin and smooth. The cut surfaces were brownish purple in color with prominent lymphoid markings.

Liver weighed 1213 gm. (288). It was firm with a thin, smooth, glistening capsule. The inferior edge was blunted with a deep prominent notch just medial to the gallbladder fossa. Both external and moist cut surfaces were pale yellow in color, and from the latter a grayish fluid could be expressed. The usual markings were obscured.

Kidneys.—The right kidney weighed 77 gm. (36); the left, 67 gm. (35). They were moderately firm. The capsules were thin and stripped readily, exposing smooth, pale pink, external surfaces. The cut surfaces were pale with well-defined markings, however.

Thymus weighed 22 gm. It was large, soft and pulpy, grayish white in color with little interlobular connective tissue.

The mesenteric, mediastinal and pulmonary hilar lymph nodes were large softish and pale yellowish gray in color. Heart weighed 50 gm. (64); pancreas and adrenals (combined weight), 9 gm.; gastrointestinal tract, bone and spinal cord were grossly not remarkable.

Only sections of liver, kidney, spleen, thymus and spinal cord were fixed in absolute alcohol at the time of autopsy.

Microscopic Examination

Lung.—There was an extensive pneumonia. The exudate varied from serous to frankly purulent with hyaline membranes in one subpleural area. Many gram-positive lancet-shaped diplococci were present within the polymorphonuclears and monocytes of the exudate. The bronchioles were variously involved, with ulceration, vascular thrombosis, and hemorrhage in some, as well as purulent exudate in the lumina of many. The fibers of their muscular coats appeared clear and vacuolated. The pleura, interlobular septums and subendothelial venous walls were mildly involved in the inflammatory process.

Spleen.—There were the same large pale cells as in the previous case, but far fewer in number. Here, too, they were histochemically inert. There was mild passive congestion with less endothelial hypertrophy. The pulp cords were cellular, chiefly due to lymphocytes. There were occasional actively phagocytic histiocytes but in all with little recognizable hemosiderin. There were large "toxic" reactive foci in the corpuscles. There were Best-positive granules in many of the polymorphonuclears and in eosinophilic myelocytes.

Pancreas.—The islets were abundant and of moderate size. There were only rare isletal cells with large nuclei, but there were many irregularly polyhedral or pyramidal atrophic forms. The cytoplasm of these was deeply acidophilic and their nuclei were dark staining, small, pleomorphic, and pyknotic. The parenchyma was not remarkable.

Liver (Figs. 6 and 7).—These sections were almost identical with those of the former case, with the following differences: there was more portal connective tissue, tending at times to outline lobules and occasionally surrounding islands of liver cells. The latter were a little more uniform and smaller in size with, in all, fewer large vacuolated cells. The cytoplasm of the smaller cells, however, contained many discrete small fatty droplets with less tendency to become confluent than in the previous case. Comparing the sections of both cases, one gained the impression that there was about the same amount of stainable glycogen but slightly less fat

in this case. The liver in this case was, however, much larger than that in the former, so that quantitatively there possibly was more fat in the liver as a whole. The same probably applies for glycogen.

Kidney.—It was the same as in the previous case except for more frequent vacuolated nuclei, containing glycogen in the cells of the convoluted tubules, larger granules of glycogen in the cytoplasm of the cells, a lesser amount of stainable fat, and no evidence of calcification.

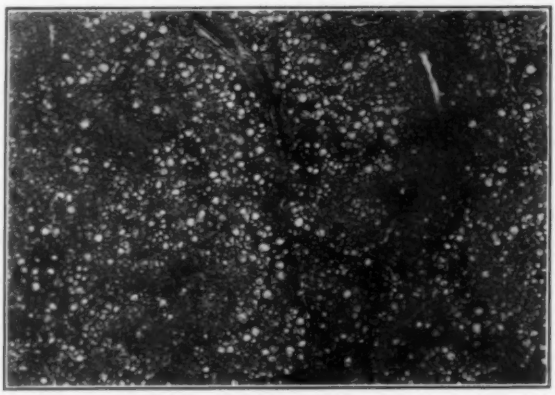

Fig. 6.—Liver (Best). There is considerable glycogen but also many large vacuolated cells. (×80.)

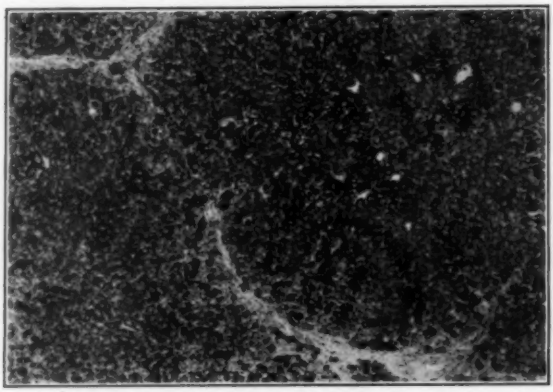

Fig. 7.—Liver (scharlach R). Note the large amount of stainable fat. An average field in a number of sections from several blocks. (×80.)

Adrenals.—The sections were identical with those of the former case particularly with reference to the degree of lipoid depletion and its distribution. There were fewer histiocytes in the medulla and the sympathochromaffin elements were more abundant.

Thymus.—Here again there was practically no involution but persistence of myelopoiesis. There were Best-positive granules in the polymorphonuclears infiltrating necrotic Hassall's corpuscles and in some of the myelocytes.

Lymph Nodes.—In those from the mesentery there were dilated sinusoids containing lymphocytes, monocytes and polymorphonuclears. In the cortical follicles there were large reactive foci, like those in the corpuscles of the spleen. The pulmonary hilar nodes presented more acute inflammatory changes.

Osseous System.—The sections of rib and vertebrae were identical with those of the former case except that the large pale cells were scarce in the rib but plentiful in the vertebrae.

Of the other sections there were chronic inflammatory changes in the esophagus with early mild ulceration and congestion of the small intestine with intramucosal lymphoid follicles containing "toxic" reactive foci. Heart, testes, and spinal cord were not remarkable. The adipose tissue was more abundant than in the former case, but there were areas of early atrophy in some of the larger fatty lobules, and in general the fat cells themselves were smallish.

Anatomical Diagnoses.—Glycogenic infiltration of liver and kidney; marked fatty infiltration of the liver; probably lipoid-staining cells in spleen and bone marrow; bronchopneumonia, marked; "toxic" reactive changes in lymphoid tissues generally; chronic passive congestion of the spleen; chronic esophagitis; lipoid depletion of the adrenal.

A third case will be described briefly, in which liver, skin, and muscle were obtained by biopsy, but in which postmortem examination was refused.

CASE 3.—E. P., aged eight years two months, had been noted to have an enlarged abdomen ever since six months of age. One sister died at the age of four years with apparently a similar condition. There was another younger sister living and well. This patient had been seen in the Out-Patient Department of this hospital on several occasions from the time she was one and two-thirds years old. Her physical development was tardy, and at the present hospital admission she appeared to be no more than five years old. The liver was always greatly enlarged. The spleen was not palpable. She had never been jaundiced. She had occasional epistaxis. For the past year she complained of fatigue. Three weeks before admission she developed extreme dyspnea and anorexia. The pertinent physical and laboratory findings were: delicate slender extremities, meager subcutaneous tissues elsewhere although full cheeks, dullness at the bases of both lungs with many moist râles; markedly protuberant abdomen with a palpable liver 10 cm. beyond the midline to the left and well below the iliac crest on the right. There was acetonuria on two occasions, red blood cells 2,520,000 with 29 per cent hemoglobin, white blood cells 7,750 with 50 per cent polymorphs, 11 per cent lymphocytes, 22 per cent young forms, and 14 per cent macrocytes. Fasting blood sugar levels varied from 51 to 72 mg. per cent. There was practically no response to adrenalin. The glucose tolerance curve rose from 72 to 166 after two hours and fell to 85 on the third hour. Serum calcium was 10.2 and phosphorus, 2.3. The nonprotein nitrogen was 18 and cholesterol, 184. She ran a constantly elevated temperature and died within a month of admission.

Biopsy Examination

Liver.—It was practically identical with that of the second case. There was, however, mild lymphocytic and polymorphonuclear infiltration of the portal areas. There was a large amount of glycogen and also fat. The latter was present in the form of smaller droplets, with, in frequent areas, larger cells with large globular masses of fat.

Skin.—There was mild perivascular lymphocytic infiltration with histiocytic proliferation. The endothelial cells of the small blood vessels were prominent. There

was mild edema of the papillary layer of the corium. The coil glands were dilated. The smooth muscles of the subcutaneous arteries were enlarged and vacuolated. The subcutaneous fat was well developed. There was no stainable glycogen in the epidermis, but this substance was abundant in the epithelium of the hair shafts, the smooth muscle of the subcutaneous arteries, and mildly in the epithelium of the ducts of the sudoriferous glands, the fat cells of one small atrophic lobule, occasional polymorphonuclears, and rare fibrocytes or monocytes.

Muscle.—There was mild infiltration of the muscle fibers with glycogen.

In spite of several months of fixation in Pick's solution (ii), the liver and kidneys of the first case contained respectively 3.04 per cent and 11.5 per cent of glycogen in terms of dry weight.[12] This compares favorably with the findings of others in this disease, due consideration being paid to the duration of fixation. In von Gierke's first case, there was 33.72 per cent glycogen for dry weight of liver and 36.82 per cent for the kidney, fresh material being employed for chemical analysis (twenty-four hours postmortem). Kimmelstiel reported 16.5 per cent for wet weight of liver after five days in the ice chest and Antopol and his coworkers, 3.25 per cent and 4.34 per cent for liver and kidney, respectively, in formalin-fixed material four weeks after autopsy. Faber found 15.3 per cent glycogen for wet weight of liver and 5.9 per cent for the kidney twenty-eight hours postmortem. Popper and Wozasek[13] give the following values for glycogen in man: with slow agonal death 0.3 to 1.2 per cent for the liver; in sudden deaths, 2.42 to 6.17 per cent. The kidneys ordinarily contained 0.29 to 0.69 per cent rising to 0.97 and 1.64 per cent in diabetics. They reported elevated glycogen values for the liver in children with diabetes and alimentary intoxication. It is obvious that the values for the two organs here exceed by far those of the normal range.

Even more striking was the finding of a total fat content of 52.3 per cent for the dry weight of liver.[14] There are very few reported analyses for the fat content of this organ in different diseases. Hoppe-Seyler's figures[15] are given in terms of dry weight in the following:

Accidental death by drowning	9.6%
Passive congestion	2.8%
Acute yellow atrophy	27.4%
Acute yellow atrophy with mild cirrhosis	9.5%
Atrophic cirrhosis	7.3 — 27.9%
Atrophic cirrhosis with diabetes	7.3%
Large cirrhotic fatty livers (Zirrhotische Fettleber)	50.3 — 74.4%
Hypertrophic cirrhosis	46.7%

The ages of the individuals from whom these were obtained varied from twenty-three to sixty-two years. In reviewing the weights of frankly fatty livers from the autopsy files of this hospital, it was of interest that a number fell below the given average, that many were

within or slightly above the average, with relatively few approaching double the normal weight. A very large percentage of these occurred in infants with nutritional disturbances, in whom, depending upon the duration of the disease and accompanying marasmus, there was in general goodly visceral atrophy. This might account for the above observations where with massive fatty infiltration there was for the most part an inappreciable elevation in weight of the liver. It is regrettable that there are no known fat values for these, but, even if one assumes that the percentage for the dry weight of them approximated that of the large cirrhotic fatty livers, there probably would be less total fat than in an organ some three and one-half times normal weight. Whether the fat factor here, even as in the enlarged livers of juvenile diabetics, contributes in any way to the hypertrophy of the organ is quite uncertain. In view of the antagonistic hydration tendencies of glycogen and fat, the former being high, the latter low, it might be expected that the fat in cells filled with stable glycogen would tend in all to be of finer dispersed form. Hence from histologic observations alone, the fat factor may appear to be inappreciable. One, therefore, gains the impression that with future chemical analyses the livers in this disease will be found to contain a considerable amount of fat, even exceeding at times those of the above mentioned type in which almost all the liver cells are stuffed with lipoids and the appearance is almost one of adipose tissue.

Associated with the marked degree of fatty infiltration of the liver also noted in the two other cases in which there were no chemical analyses, there were, clinically, a distinct lipemia, with a serum cholesterol of 394 mg. per cent, acidosis with a carbon dioxide combining power of 24 volumes per cent and acetonuria. Another unusual morphologic finding was the presence of large vacuolated cells in the spleen and bone marrow of both this and the second case. These failed to stain for fat or glycogen, although various methods were employed. Yet there is no reason to believe that they contained glycogen in view of the apparent stability of this substance in this disease and therefore little chance of complete lysis having occurred here and not elsewhere, as well as the fact that the leucocytes in the same organ contained granules which stained readily with Best's stain and iodine. There is far more reason to believe that they stored lipoids. Morphologically they resembled fat-laden monocytes without, however, any of the characteristics of the Gaucher cell. Furthermore, Warren[16] states that "aside from this fairly common finding of rare lipoid-containing cells in the spleen, there are a few cases of diabetes with lipemia recorded, in which very marked hyperplasia of these cells occurs, and the lipoid present does not as a rule give the staining reactions usually en-

countered." It is possible that here one is dealing with a complex combination of fats of which poorly stainable or nonstainable chemical forms may be present.

This leads one to draw an analogy between this disease and diabetes mellitus, at least from the point of view of fat metabolism. If it is true, in a physiologic sense, that diabetes is characterized by excessive, and this disease by diminished, glycogenolysis, then in both, fats will be excessively burnt, particularly when there is greater metabolic activity. In both, therefore, there may be lipemia and ketosis. But, whereas in diabetes there are hyperglycemia and glycosuria, here there are hypoglycemia and no glycosuria. Furthermore, enlarged fatty livers in juvenile diabetics, particularly, are not at all infrequent, and in instances with lipemia, they too have had fat-laden monocytes in the spleen.

In the review of the histologic observations of fat in the liver reported in the literature in this disease and cited elsewhere in this paper, as well as in the present cases, it is obvious that the amount of fat in the organ can be quite variable. Recently there have appeared several reports dealing with hypertrophy of the liver in infancy, said to be due to massive fatty infiltration. Bjorum[17] in 1926 reported in the same family four cases with greatly enlarged livers, which he diagnosed as adipositas hepatis, at postmortem. Debre and Semelaigne[18, 19] have placed four of their living cases of hepatomegaly into the same category, on the basis of a single biopsy from one. The clinical aspects of their cases agree favorably with those of glycogen disease. Van Creveld[20] has emphasized that variations in clinical behavior and laboratory findings are to a degree to be expected. In both these reports there were no histochemical studies for glycogen. It is to be emphasized that fat and glycogen may coexist abundantly in the same organ and in the same cell. There is, however, one puzzling report by Kramer, Grayzel, and Solomon,[21] which appears to stand alone in this respect. It is that of a child, eight months of age, with hepatomegaly, marked hypoglycemia, no response to adrenalin, and no acetonuria. There was extensive fat replacement of the liver and with Best's stain no evidence of glycogen. Whether in the group of hepatic hypertrophies in infancy and childhood, excluding the cirrhoses and other well-defined lesions, and of which glycogen storage disease at present forms a well-recognized division, that of pure massive fatty infiltration alone, will also form a distinct group is not as yet entirely clear. There have been clinical descriptions of instances without anatomic studies where the disturbance in carbohydrate metabolism has been considered to be of the nature of absence or disturbed glycogenesis (*azoomylie* and *dyszoomylie* of the German literature). It may be that, in these, future pathological studies may reveal

that the accompanying enlarged livers in such instances are due to fatty infiltration alone with no abnormal glycogen storage in this or other organs.

Lastly, in the first two cases there were several features in common, viz., considerable depletion of the lipoids of the adrenal as described by Goldzieher[22] in diabetic lipemias; practically no involution of the thymus with, however, persistent myelopoiesis; evident lines of arrest of bony growth characterized histologically by the persistence of transverse bony trabeculae; and some degree of atrophy of the adipose tissues in general.

CONCLUSIONS

Two cases of glycogen storage disease with autopsy findings and one with biopsy of the liver are described.

Disturbances in fat metabolism, accompanying this disease, are stressed, and an analogy is drawn between glycogen storage disease and diabetes mellitus from that standpoint.

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ANGIOMAS IN PREMATURE INFANTS

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TUMORS of the blood vessels are the most common type of neoplasm occurring in infancy. This communication deals only with angiomas of the skin in premature infants seen in the premature clinic of the Sarah Morris Hospital in the three-year period, 1932 to 1935. Although these lesions are by no means rare in either full-term or premature infants, their incidence according to the author's knowledge has not been previously computed. Of 344 premature white infants observed during this period, 40, or 11.64 per cent, were so affected. In 78 premature negro infants seen during the same period, no angiomas were noted.

VARIETIES OF TUMORS AND TIME OF APPEARANCE

Thirty-five of the infants had angioma simplex, so-called strawberry marks; 4 had cavernous angiomas; and 1 had a nevus flammeus, or port-wine stain. These diagnoses are clinical, based only on their appearance, as it is impossible to classify accurately the character of the tumor without histologic examination. In only 7 instances were these marks noted at birth, and in each case the lesion developed into a comparatively large tumor. The nevus flammeus noted was also present at birth. In all of the other instances, the lesion first made its appearance between the first week and the second month of life. The angiomas first appear as slight irregular discolorations, of the skin, which rapidly enlarge and become elevated until they assume their characteristic red spongy appearance. According to Bland-Sutton,[1] an angioma may be present at birth, but much more frequently it appears in the first few weeks of life. Fitzwilliams[2] stated that this was a widely believed fallacy, and he presented figures in 645 cases showing 537, or 83.2 per cent, of the lesions present at birth. His investigation showed that the marks were usually noted at the twelfth to fourteenth day of life and concluded that this was the first day that the mother bathed the baby and thus saw it undressed for the first time. He therefore felt that the figure of 83.2 per cent underestimated the true facts, and that practically all vascular nevi were present at birth. Culver,[3] in reporting a series of 23 infants with angiomas, found that 14 were present at birth. Other authors agree that the lesions are either present at birth or may appear at any time up to the first several months of life.

In the 40 infants in whom lesions were found, there were 60 angiomas noted. Twelve had multiple lesions, 7 being the greatest number found

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in any one infant. No convulsions, spastic hemiplegias, or mental deficiency which might be referable to associated angiomas of the brain have occurred.[4]

LOCATION OF TUMORS

Scalp	3	15	25.0%
Face	8		
Neck	4		
Upper extremities	12	19	31.6%
Lower extremities	7		
Anterior thorax	7	26	43.3%
Back	9		
Abdomen	8		
Genitals	2		

These tumors differ in location from the usual sites reported by other observers. In this series, 15 (25 per cent) were on the head and neck;

Fig. 1.—A. S., aged six weeks. Angioma simplex at side of nose first appeared at the age of nineteen days.

19 (31.6 per cent) were on the extremities; and 26 (43.2 per cent) were on the trunk or genitals. In Fitzwilliams' series[2] of 645 cases, 49.3 per cent were found on the head and neck, 21.2 per cent on the extremities, and 29.2 per cent on the trunk. Taylor,[5] in reviewing all the cases treated at Huntington Memorial Hospital from 1914 to 1925, found that in the 193 patients seen, 167 lesions were on the head and neck, 19 on the extremities, and 15 on the trunk. In Rulison and McLean's series[6] of 33 patients with 43 lesions, 60.5 per cent were on the head; 18.6 per cent were on the extremities; and 20.3 per cent were on the trunk. In Culver's series[3] of 29 angiomas in 23 infants, there were 25 about the head, mostly on the face. In Robinson's series[7] of 276 infants with 297 angiomas, there were 76 per cent on the head and neck, 13.1 per cent on the extremities, and 10.8 per cent on the trunk. Geschickter[8] noted that the distribution of 370 cutaneous angiomas was as follows: head and neck, 201; extremities, 110; trunk and genitals, 59. He stated that

his study included only lesions based on surgical and autopsy records and did not consider the very common angiomas on the surface of the body which escaped clinical and pathological study. MacCollum[9] recently reviewed 697 angiomas seen at Children's Hospital in Boston and noted that 469 (67 per cent) were on the head and neck.

The above statistics were compiled mainly by consultants to whom these patients were referred for treatment. This fact probably accounts for the prevailing statement in the literature that angiomas predominate on the head and neck. Since angiomas in other locations are not as objectionable from an esthetic viewpoint, they do not reach the attention of a consultant as commonly, and hence are not included in these authors' statistics.

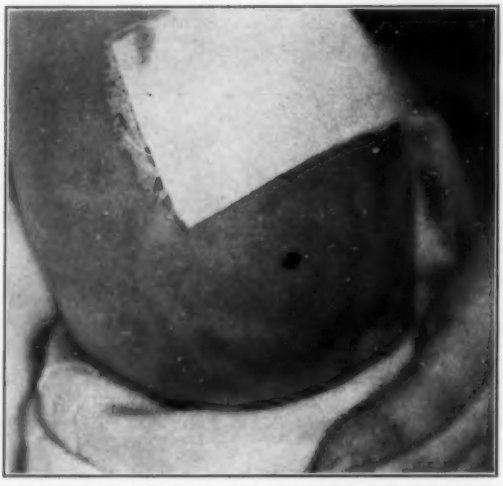

Fig. 2.—B. T., aged six weeks. Angioma simplex on abdomen first appeared at the age of twenty days.

RELATION TO SEX

Twenty-six of the infants (65 per cent) were female, and 14 (35 per cent) were males. These figures correspond exactly with those of Fitzwilliams,[2] who found 65.2 per cent in females and 34.7 per cent in males. In Robinson's series[7] there were 68.1 per cent females and 31.9 per cent males. MacCollum[9] found 69 per cent females and 31 per cent males. This constitutes a rather striking similarity of figures in both large and small groups of infants.

RELATION TO BIRTH WEIGHT

In an incomplete review of the same group of premature infants which I am reporting, Mohr[10] previously noted that angiomas and other nevi are more common among the prematurely born than among their sib-

lings. He stated that in this group these conditions were observed to occur only among the children with low birth weights, none of the infants having weighed as much as 2,000 gm. at birth. A study of the birth weight of the 40 infants making up this series shows that 9 infants weighed between 1,000 and 1,500 gm. at birth, 19 weighed between 1,500 and 2,000 gm., and 12 weighed over 2,000 gm.

EMBRYOLOGY AND HISTOPATHOLOGY

The primitive circulatory system develops from the mesoderm overlying the yolk sac at the junction of the foregut and hindgut. Blood islands, isolated areas of formative tissue, occur here and by joining to-

Fig. 3.—M. M., aged seven weeks. Angioma simplex on back first appeared at the age of twenty-three days.

gether develop a network of vessels over the yolk sac. From this mesenchymal network the circulatory system grows by extension. Proliferative growth of the endothelial lining links the vascular spaces into continuous channels, which further expand by independent sprouting.

Three clinical forms of angiomas are recognized: 1. The simple raised type, angioma simplex, or strawberry mark, is composed of endothelial lined spaces of varying sizes. Both venules and arterioles may form the constituents. There appears to be an endothelial proliferation of the intima in the form of outbuddings of a parent vessel. Histologically these vessels are separated by a narrow strip of connective tissue, or are freely connected with each other by a system of vessels.

2. Cavernous angiomas are deep seated. They are either venous or arterial in origin, and are usually deep purple in color, having the same structure as erectile tissue. Histologically they contain larger vessels than the angioma simplex.

3. Nevus flammeus, or port-wine stain, usually occurs in the upper layer of the corium. Here are formed a large number of dilated capillaries which are distinctly elongated and extend in various directions. These are particularly of the venous type.

THEORIES OF CAUSATION

Unna[11] believed that abnormal pressure during fetal life was the genetic factor in the production of angiomas. Ribbert's theory[12] was that these tumors develop from embryologic rudiments. Virchow[13] believed that these tumors result from the action of local irritation on imperfectly formed vessels, as those found in embryonal fissures. According to Ewing,[14] their origin lies in a developmental anomaly in the structure of certain vascular segments which retain embryologic characters. He states that the congenital origin of the great majority of angiomas speaks strongly in favor of a tissue predisposition as a prominent factor in their genesis. Some authors[2, 15, 16] believe that there is a relationship of these lesions to the distribution of the peripheral nerves. Laidlaw and Murray[17] have extended the theory that angiomas may be phylogenetic remnants of early vascular tufts that served as accessory lung tissue in the skin of reptiles and lower forms. Their occurrence in man might then be compared with the appearance and disappearance of the gill arches, suggesting that such tumors may be the remains of the vascular tufts that did not disappear in early embryonic life.

Because of the greater incidence of these lesions in premature infants, the author suggests that there is a relationship between the production of angiomas and the peripheral vascular needs of the body. Thus, these lesions appear at a time of rapid growth, when the body surface is expanding and has a demand for increased blood supply. During part of this period the temperature mechanism of the premature infant is poorly regulated, and the skin temperature is lower than normal. The blood pressure at this time is increasing with the growth of the infant.[18] Thoma[19] has stated that increase of blood pressure tends to excite new growth of blood vessels, although Leader[20] believes that this would cause an increase in the size of vessels already present rather than overproduction.

COURSE AND TREATMENT

With the exception of the nevus flammeus and small vascular papules that remain stationary in size, all of these angiomas have shown signs of rapid growth immediately following their appearance. In a few of these infants who were untreated for various reasons, the growth persisted until about the age of six months, when its maximum size was

reached. Seven infants had lesions so small that no treatment was deemed necessary. Five infants failed to return for observation. In four infants with moderately large lesions spontaneous regression after six months has occurred. Twenty-four patients have received treatment by exposure to radium.

The optimum time to begin treatment and the type of treatment advised have long been subjects of considerable controversy. Many forms of therapy have been reported with varying success, mainly radium, x-ray, freezing with carbon dioxide snow, removal by surgery, cautery, injection of various substances from boiling water to sodium morrhuate, chemical caustics, etc.

Traub[21] recently reviewed the subject of whether vascular nevi should be treated, by questioning 117 members of the American Dermatologic Association. Sixty-three per cent of the answers indicated that the authors had seen an infrequent spontaneous disappearance (1 to 2 cases each). From the answers he concluded that it is best to wait for a very short time only in stationary angiomas, but in progressive lesions immediate measures are advisable. He further states that today, probably more men prefer radium than any other method of treatment. Culver[3] states that a baby can receive treatment at any age with complete safety. Simpson[22] believes that treatment should be begun in earliest infancy. Rulison and McLean[6] conclude that the younger the patient the better and more rapid are the results under radium therapy. Schamberg and Bradley[23] were of the same opinion and stated that their best results were obtained in infants in whom treatment was started between the fourth and tenth week. Authors advising other types of treatment, namely, x-ray and carbon dioxide snow, believe that treatment should be started as early as possible. MacCollum stresses the use of endothermy coagulation in raised lesions, emphasizing, however, that in many instances no one method is sufficient.

In this group of infants radium* has been applied at the age of three to six months, although I believe that it may be used earlier with safety. The cavernous angiomas have been treated by means of interstitial radiation. Monel needles containing platinum cells are inserted for periods varying from twenty-four to forty-eight hours. The other angiomas have been treated by means of external radiation with radium plaques. The treatments have been administered in divided doses. From 4 to 12 applications at one-month intervals have been necessary, depending upon the size of the lesion. In this manner local reactions are completely eliminated.

The response of the lesion to such treatment is slow and occurs over a period of months. It is believed that this form of regression represents

*Radium therapy was administered by the tumor clinic under the direction of Dr. Max Cutler.

the ideal in the treatment of these lesions, and it is the purpose of the technic employed to cause a slow regression without violent reactions in normal tissues.

CONCLUSIONS

1. Of 344 premature white infants 40 (11.64 per cent) developed angiomas. In 78 premature negro infants no angiomas occurred.

2. Thirty-five of the infants had angioma simplex, 4 cavernous angiomas, and 1 nevus flammeus.

3. Fifteen (25 per cent) of the lesions occurred on the head and neck, 19 (31.6 per cent) on the extremities, and 26 (43.3 per cent) on the trunk; 26 (65 per cent) of the infants affected were females, and 14 (35 per cent) were males.

4. It is suggested that there is a relationship in the development of the angiomas to the peripheral vascular needs of the infant.

5. Radium has been used successfully in the eradication of these lesions with no local or general reactions.

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SODIUM CHLORIDE IN THE TREATMENT OF NOCTURNAL ENURESIS IN CHILDREN

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THE treatment of nocturnal enuresis in children has been a rather desultory and haphazard procedure. In the management of a case one method after another has usually been tried in the hope of finding something eventually that would control the bed-wetting. From the physical standpoint, perhaps the most satisfactory results have been obtained by the limitation of the fluid intake toward the end of the day in conjunction with the administration of some form of atropine. From the mental aspect, the condition not infrequently has responded to the simple use of suggestion or to the more complex analytic approach. But in many instances, all measures have proved unsuccessful.

Quite by accident, Krasnogorski[1] came upon a new and interesting idea for the control of this condition. In 1927 he and his coworkers, Gantt and Harbedia, were engaged in experimental work upon the salivary glands. They were trying to determine what factors influenced the quantity of secretion from these bodies. Neither the elimination of water from the diet nor the production of a profuse perspiration by sun baths decreased the amount of secretion except in one group of children. At first this seemed puzzling, but further investigation disclosed that this group in whom water was eliminated from the diet happened to have had a breakfast which included salt ham and herring. This suggested a possible association of the salty diet with the diminution of the secretion. Subsequent experimental work along this line revealed that sodium chloride if given in sufficient amounts invariably reduced the amount of salivary flow. Later this lessening of secretion by an increased salt intake was even more strikingly demonstrated in relation to the pancreatic juice.

Krasnogorski then decided to apply this principle to the secretion of urine in cases of nocturnal enuresis in children. The salt was given in the form of a caviar or ham sandwich when the child was already in bed and about ready to go to sleep. This arrangement left no time for the development of thirst while awake. Children can sleep all night in spite of a slight or moderate thirst. In addition, the routine care included the elimination of fluids after four o'clock in the afternoon and a strictly dry supper. Under this plan of procedure, Krasnogorski reported consistently good results.

From the Department of Pediatrics, Sea View Hospital, New York, and private practice. Read before the Metropolitan Medical Society, New York, Dec. 11, 1935.

About two and one-half years ago at the suggestion of Dr. Béla Schick, we decided to try this method on two groups of children suffering from nocturnal enuresis. One of us (Rosenson) planned to follow the results of its application in private practice; the other (Liswood), in an institution. The pediatric service at Sea View Hospital was well adapted for this latter study.

Sea View Hospital is an institution devoted to the care of tuberculous patients and affords ample opportunity for long periods of observation. Among the children there is the usual quota of enuretic cases found in any child-caring institution. The patients selected for the study were ambulatory cases showing no evidence of open tuberculosis. There were eighteen such cases with nocturnal enuresis. Their ages ranged between four and seven years. There were eleven boys and seven girls. Physical examination and repeated urine tests failed to reveal an organic basis for the enuresis in any of the cases.

Before inaugurating the new treatment, a control period of a week during which all fluids were withheld from the children's diet after 4 P.M. was instituted. The mere elimination of liquids in this way did not seem to control the enuresis in any of the cases. No other treatment for the bed-wetting as far as is known had been previously tried on any of these hospital patients.

The cases observed in private practice were for the most part brought for the treatment of the annoying and troublesome bed-wetting. Ten children were included in this group. As far as could be determined by physical examination and urinalysis, the nocturnal enuresis in these cases, too, was functional in character. The youngest child was four and the oldest ten years. There were six girls and four boys.

Various methods had been tried among this private group of children. The curtailment of liquids before retiring, the administration of tincture of belladonna, awakening of the child at intervals to urinate during the night, the psychologic approach including suggestion and the awarding of gold stars and prizes; all these time-honored procedures had been employed without producing any lasting successful results for the control of the nocturnal enuresis.

THE DAILY REGIME FOR THE CONTROL OF NOCTURNAL ENURESIS

During the day the child is given his regular diet. Until 12 o'clock noon, he is allowed to take as much fluids as he desires; after that, fluids are somewhat limited until 4 P.M. At that time he is offered his last drink of water or milk until the following morning. For supper at 5:30 he is served with food containing as little water as possible. Such dry food includes meat, fish, egg, bread, butter, cheese and crackers. No vegetables, fruit, or cereal are offered.

When the child is already in bed for the night and all his wants and requests have been attended to, he is given the salty sandwich. To

satisfy differences of taste and to vary the monotony of repetition in food, we concocted a number of salty sandwiches. Krasnogorski found that about 5 gm. of salt are needed to obtain a therapeutic result. With some slight variations we have kept to this amount. It is important to mention here that in Krasnogorski's original publication there was a serious typographical error. The amount of salt required was given as 5 mg., when it should have been 5 gm. This was made clear to us in a personal communication.

The salt is given most effectively when combined with fat or meat abundant in binding tissue. Under such conditions the salt is absorbed gradually and the restraining influence upon the discharge of urine is prolonged. Caviar is rich in salt, but its general use here is obviously precluded. For our various sandwich fillings we used measured quantities of salt butter, ham, bacon, boned herring, boneless salt codfish, Swiss cheese, smoked salmon, and salt. To make the herring or fish more palatable, chopped hard-boiled egg or raw scraped apple was added.

The following is a list of recipes which we used for the filling of sandwiches:

SANDWICH FILLINGS	QUANTITIES	GRAMS OF SALT
Bacon	6 strips	3
Salt butter	2 teaspoonfuls	0.5 to 1
Salt	¼ teaspoonful	2
Boned herring	30 gm.	5
Salt butter	2 teaspoonfuls	0.5 to 1
(Chopped hard-boiled egg)		
Ham	45 to 50 gm.	2
Swiss cheese	30 gm.	2
Salt butter	2 teaspoonfuls	1
Salt	¼ teaspoonful	2
Boneless salt codfish	35 gm.	4 to 5
(Raw scraped apple)		
Salt butter	2 teaspoonfuls	0.5 to 1
Smoked salmon	45 to 50 gm.	4 to 5
Salt butter	2 teaspoonfuls	0.5 to 1

At the end of about two months of this regime a more varied evening meal was offered. Cereal, vegetables, and some fruit were gradually added. This more liberal plan was continued for two months longer, after which the salt was slowly decreased by reducing the size of the sandwich by one-quarter at intervals of about two or three weeks until it was entirely discontinued. The length of these intervals varied in individual cases in accordance with the response of the child to the decrease in the amount of salt. If at times bed-wetting occurred on the reduction of the salt intake, it became necessary for a while to order a larger portion of salt sandwich. In most cases, the reduction of the salt intake was gradually accomplished without any recurrence of the nocturnal enuresis.

In the study of our institutional cases, the ward nurses were given detailed directions for the carrying out of the plan and were asked to

watch carefully through the night for any evidence of bed-wetting. Each such instance was charted.

RESULTS

Little difficulty was encountered in feeding the children the salty sandwiches. Their sleep was not disturbed by any undue thirst. In seventeen of the eighteen institutional cases, prompt cessation of the nocturnal enuresis occurred. In three of these cases there was a temporary relapse during the second week when two instances of bed-wetting were scored against one child and one instance against two children. From then on there followed an unbroken succession of nights with no bed-wetting. This group of cases has been followed for two years or longer and has remained free from the undesirable habit. One child failed to respond to the dietetic regime. This child, who often refused to comply with routine orders, was afflicted with epilepsy.

The cases studied in private practice were treated at home. The mothers were given full written instructions, including the list of recipes for the salty sandwich fillings. Emphasis was laid upon the importance of carrying out every detail of the plan. In the cases where complete cooperation was secured, the nocturnal enuresis promptly stopped. In two of the children a temporary lapse occurred during the third month of treatment, which was overcome by an increase in the salt intake. The treatment was entirely discontinued after six months and the children have remained free from bed-wetting up to the present time. Urine examinations showed no evidence of kidney irritation at any time.

CONCLUSIONS

Among twenty-eight cases of nocturnal enuresis treated with sodium chloride, successful results were obtained in all but one. Our inability to control the enuresis in this instance was due to the child's refusal to cooperate.

The cases followed are too few to draw any statistical conclusions. Krasnogorski, in a recent personal communication, reports 78 per cent of successful results among 125 cases treated.

Our study suggests that nocturnal enuresis in children can be controlled, in a large proportion of cases, by the dietetic regime described. However, before applying this regime in any given cases, an effort should be made first to find and treat any underlying physical or psychologic condition that might account for the bed-wetting. If such attempts fail, the sodium chloride treatment, as outlined, offers an effective method.

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THE CONTINUOUS INTRAVENOUS METHOD OF SERUM ADMINISTRATION

Case Reports

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FOR many years the continuous intravenous injection of 5 per cent glucose in physiologic saline in the treatment of infants and children with toxemia or infections involving the meninges has been used extensively at the Hospital for Sick Children. The administration of large doses of antitoxin in the treatment of tetanus[1] and meningococcic infections has been carried out by injecting the therapeutic antiserum by means of the continuous intravenous apparatus with the glucose-saline solution. The method employed, with a diagram, is described in detail by Brown and Tisdall.[2] Results of the continuous intravenous administration of glucose saline as a therapeutic agent in the toxicosis of infants were reported by Schick and Karelitz.[3] Chargin, Leifer and Hyman[4] reported a series of cases in which large doses of neoarsphenamine were administered in a similar manner. This method is one which may with advantage be adopted in the administration of large amounts of serum to patients.

There is not infrequently some hesitation on the part of physicians in administering antitoxic horse serum intravenously. In recent years various types of antitoxin have, nevertheless, been injected into the veins of critically ill patients suffering from a variety of diseases. The serum is usually given by direct venipuncture. The use of foreign protein (i. e., horse serum), so given in from 15 c.c. to 50 c.c. doses, may produce a marked reaction. The patient has a rise in temperature, rapid respiration, profuse perspiration, cyanosis, and chills, a sequence of events which often causes anxiety on the part of the attending physician. Yet the intravenous method of therapy is undoubtedly the most rapid and effective way of neutralizing toxins in the blood stream. In the present communication a method is described for the administration of antitoxic horse serum which we feel materially reduces the incidence of immediate serum reactions. A summary of the different types of disease which have been treated and the final outcome will be outlined.

METHOD OF THE INTRAVENOUS USE OF ANTITOXIN

The usual apparatus for the continuous intravenous administration of glucose saline is set up with the gold cannula tied in the internal saphenous vein of the leg. The rubber tubing above the drip bulb is

From the wards and laboratories of the Hospital for Sick Children, the Department of Paediatrics and the Connaught Laboratories, University of Toronto.

well scrubbed with an alcohol sponge, and the antitoxin to be administered is injected by needle and syringe at this site. The first dose thus introduced is 0.5 c.c. This is followed in from one-half to one hour with 1 c.c. and increased by 1 c.c. each hour until a total of 5 c.c. at hourly intervals is given. The antitoxin is given in this manner until the total dosage required is used.

The following charts summarize the case histories of patients suffering from tetanus and from streptococcic infections who were treated by this method, using tetanus antitoxin and scarlet fever antitoxin, respectively.

TABLE I

CASES OF TETANUS

NAME	AGE	ANTITOXIC UNIT PER C.C.		TOTAL AMOUNT A.T. UNITS	REACTION	RESULT
		BEFORE A.T.*	DURING A.T.			
B. J.	7 days	<0.001	-	14,000	None	Died
R. H.	11 days	-	>50	108,500	None	Recovered
D. C.	6 yr.	-	-	1,150,000	None	Recovered
H. M.	12 yr.	<0.001	>25	9,000	None	Recovered
J. H.	3½ yr.	<0.001	>75	214,000	None	Recovered
G. B.	12 yr.	-	-	135,812	None	Recovered

*Antitoxin.

It will be noted that in cases of tetanus in which samples of blood were taken, the antitoxic unit per cubic centimeter was negligible before the use of serum and was raised to from 25 to 75 units per cubic centimeter after antitoxin administration. In all of these patients no immediate reaction to serum occurred although in certain instances very large doses were administered. An infant, eleven days old, made a complete recovery after the administration of 108,500 units of tetanus antitoxin.

TABLE II

CASES OF STREPTOCOCCIC INFECTIONS

NAME	AGE (YEARS)	TYPE	TOTAL AMOUNT A.T.* INTRAVENOUSLY	REACTION	RESULT
G. G.	7	Meningitis	188 c.c.	None	Died
R. G.	8	Meningitis	155 c.c.	None	Died
S. A.	2½	Meningitis and septicemia	164 c.c.	None	Died
J. S.	7	Meningitis	100 c.c.	None	Died
M. M.	7	Meningitis	101 c.c.	None	Died
M. J.	13	Meningitis and septicemia	270 c.c.	None	Died
W. T.	7	Septicemia	150 c.c.	Febrile	Recovered
K. McC.	10	Septicemia	70 c.c.	None	Recovered

*Antitoxin.

From Table II it will be observed that although there were no recoveries with the intravenous or intrathecal use of scarlet fever antitoxin in cases of meningitis, no reaction to serum occurred. Most of

these children died as a result of meningitis some few days after antitoxin administration had been discontinued. It is also noteworthy that two patients with septicemia made a complete recovery. In one of these children several forms of treatment had been tried for a week before the administration of antitoxin.

DISCUSSION

In the series of cases reported there have been spectacular results as well as failures, which are inevitable, with present methods of prevention and treatment. The observation was that the majority of these patients had no immediate direct reaction to serum. In one instance sensitivity to horse serum was noted by the presence of an urticarial-erythematous reaction in the skin. This patient received horse serum intravenously without the slightest sign of systemic reaction. Often, not even a febrile response was noted, although many of the patients had some fever with their infection. In one instance the child was admitted in a critical condition. His temperature was 107° F. He was pulseless, cyanosed, and in a moribund condition. A continuous intravenous injection was commenced immediately, and scarlet fever antitoxin was given by this method. In spite of the septicemia and meningitis due to the *Streptococcus hemolyticus* he lived for nine days after admission, and his temperature remained at a lower level than before the administration of antitoxin. It is noteworthy that there was no difference in the incidence of serum disease from that usually found after the use of horse serum by other methods.

In conclusion, the author believes that a reasonably effective method of administering antitoxic horse serum to infants and children, which produces minimal or no reaction, has been outlined. Large quantities of serum well diluted with glucose in normal saline may be given within very few hours by use of the continuous intravenous method. This method of therapy has been applied to a variety of clinical conditions for a period of two years with no general reaction following the use of large doses of antitoxic horse serum intravenously.

The author is indebted to Dr. E. G. Kerslake for blood antitoxin studies.

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PUBERTY MASTITIS

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WITH the increase of public education in the field of cancer and allied medical subjects, it is my impression that parents are often unnecessarily alarmed over a condition that occurs in children at or just before puberty, frequently in girls but not infrequently in boys. This is a tumorlike enlargement of one or both breasts known as *mastitis adolescentium* or puberty mastitis.

While this condition is familiar to pediatricians, the general physician is not as well acquainted with it as he should be to prevent often unnecessary operations and much needless worry. There are very few references to the condition; practically none in the pediatric literature, and the few descriptions in textbooks on pathology and surgery are rather indefinite and at variance with each other, so that there seems to be no unanimity of opinion about it.

The very scanty reference to this more or less physiologic process was noted by Still, who quotes Underwood as giving the first description of mastitis occurring in children at a variable time before puberty. A condition, which Still remarks, is so little known that he has more than once known operations proposed for it because of the mistaken idea that it was a malignant growth. In Underwood's early description, "Induration of the Breasts," he says: "It would be improper to pass over the slightest affection that has on many occasions exceedingly alarmed parents and sometimes perplexed the younger part of the profession. Of this kind, are affections of the breasts in female children previously to, or about the time of, their beginning to enlarge. At this period they sometimes become very painful and upon examination a hardness and swelling are discovered and in some instances sharp points may be felt, which are very painful when pressed. The hardness is deep-seated, around and behind the nipple and is sometimes loose and at other times fixed and attended with severe lancinating pains which have given rise to disagreeable suspicions in regard to the probable nature of the complaint, and would indeed at a more advanced age claim serious attention. When happening in families addicted to scrofula, that disorder is naturally suspected, and sometimes a morbid scirrhus has been feared."

Underwood further states: "In some instances only one of the breasts is affected and after some months the other or sometimes both nearly at the same time. Yet no evil consequence in any instance under my eye has

From the Sarah Morris Children's Hospital of the Michael Reese Hospital.

ever ensued in patients at the above-mentioned age nor have I heard of any from other practitioners though it has sometimes been seriously apprehended."

This gives a fairly clear picture of the condition, and is more accurate than the few references I have seen in modern textbooks.

Some eighteen or twenty years ago I saw my first patient with this condition. The mother was very much alarmed about the swelling of her child's breast just behind the nipple. I could find practically no reference to the condition in any pediatric text. Since then I have seen, like all pediatricians, many children with similar swellings.

Two years ago I was prompted to write this paper when a mother brought her ten-year-old daughter to me with a story that she had a breast removed six months previously and now had, she was sure, a similar swelling in the other breast. I found it a typical case of mastitis adolescentium.

The breast is a modified sudoriferous gland and physiologically is related rather intricately to the reproductive system. The epithelial lining of the ducts and alveoli is derived from the ectoderm and the supporting connective tissue from the mesoderm.

Each breast consists of fifteen to twenty lobes, which are themselves branched saccular glands whose lactiferous ducts open on the surface of the nipple near its apex. The main lactiferous ducts subdivide into many interlobular ducts about which are clustered the group of secreting acini. Each group forms one of the many lobules included in one lobe of the gland.

At puberty, and again at the end of pregnancy, marked enlargement of the gland and the development of additional lobules and acini take place. There is also an increase in the deposit of adipose tissue at puberty.

Goltz and Evald have shown that the development of the mammary glands at puberty is due to the action of ovarian hormones, both follicular and luteal. C. W. Lurner has demonstrated that the follicular hormone stimulates the development of the duct system, and the corpus luteum the alveoli. The anterior pituitary gland is also active and important in the production of lactation (Stueker and Grueter).

Cheatle and Cutler[2] regard puberty mastitis as a result of some aberrance in the physiologic hyperplasia of the mammary tissues, probably an overactivity of the tissues in response to an excess of growth-stimulating substances.

Muir[3] speaks of mastitis adolescentium as a nonsuppurative acute inflammatory condition similar to puerperal mastitis.

Rose and Carless[4] write of mastitis adolescentium as being a condition similar to mastitis neonatorum, which it does resemble very closely.

Kaufmann[5] states that puberty mastitis is probably the result of irritation of the ducts due to the retention of the material secreted by the ducts. This secreted material provides a favorable medium for the growth of organisms.

Walther[6] remarks that the enlargement of the breasts in puberty very often coincides with the presence of a positive tuberculin reaction. In other words, he believes that this type of mastitis is a manifestation of a tuberculous infection. The connective tissue proliferation is explained on an allergic basis.

Ochsenius,[7] although not completely contradicting Walther, reports three cases in which the tuberculin reaction was negative.

Fig. 1.

Fig. 2.

Fig. 1.—Girl, nine years old. Swelling of left breast for one month. Typical tumorlike enlargement behind nipple. Mass completely disappeared in four months. This girl had a severe eczema and asthma.

Fig. 2.—Boy, sixteen years old. Swelling of both breasts but more marked in left. Location typically behind nipple. Complete disappearance in five months.

In none of my patients was there a positive tuberculin reaction, and I feel definitely there is no relationship. Of the ten or twelve cases I have seen in the last two or three years, five or six had a definite allergy characterized by a severe eczema, asthma, or both.

Harbeson[8] describes a case of mastitis adolescentium in which the tumor began in the upper outer quadrant of the left breast. Then two months later another tumor developed in the same location. Soon afterward, the right breast was the site of a similar tumor. The location, relative to the nipple, was not mentioned, and the last note several months later stated that the tumors were smaller but still present. They

were probably fibroadenomas. Harbeson thinks it is due to a preponderance of luteal hormone which produces an overstimulation and rapid development of the acini before development of the ducts.

The children I have seen with this tumor in the breast all had characteristics similar to those described by Underwood one hundred and fifty or more years ago. They occur more frequently between the ages of eight or nine and fifteen or sixteen years. The majority are found between the ninth and eleventh years in girls. One or both breasts may be involved, and one side may precede the other by as long as a year or more. The areola becomes darker in color and, as Cheatle and Cutler described, the lesion assumes definite and very characteristic features.

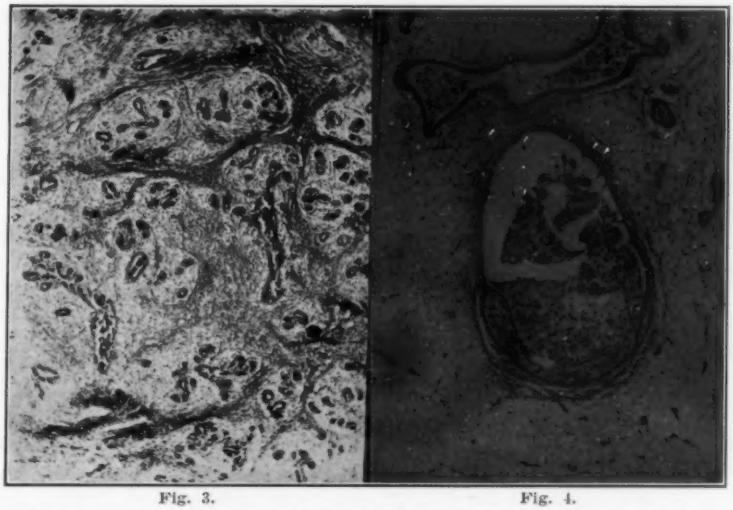

Fig. 3. Fig. 4.

Figs. 3 and 4.—Periacinous and pericanalicular infiltration and epithelial hyperplasia. (Fig. 3, ×90; Fig. 4, ×150.) (These photomicrographs were kindly lent by Dr. Max Cutler, of Michael Reese Hospital.)

The swelling forms a firm disklike or spherical tumor varying from 3 to 5 cm. in diameter, which is painful on pressure only. It is situated directly behind the nipple, which usually is flattened and often slightly inverted by the enlargement. Occasionally a few drops of an opaque serous fluid may be expressed from the nipple. The location in those I have seen is always behind the areola, and this differs from most of the other possible tumors of the mammary gland.

Puberty mastitis is not a true inflammation, but is due to an increased physiologic hypertrophy of the pericanalicular and periacinous connective tissue. The proliferative changes closely resemble those which are produced by the injection of estrin (as shown by Lacassane). It is possible that trauma may aggravate it or be responsible for its appearance

in only one breast. Usually the induration disappears as the breast develops fully. Some disappear in a few weeks while others require months.

In the male sex this condition begins at the earliest in the fourteenth or fifteenth year of life, and it may be so marked as to constitute a true fibrous tumor. Zappert is of the opinion that in the male this state is associated with an advanced development of the genital glands.

This condition is not to be confused with certain tumors which may arise only occasionally in breasts of children.

In all the children I have seen with puberty mastitis, the mothers were worried because they feared a cancer or tumor. The age, location and characteristics outlined will be sufficient readily to differentiate it from other pathologic entities.

SUMMARY

Puberty mastitis (mastitis adolescentium) has definite characteristics. It is always behind the nipple and occurs in girls between eight and twelve years of age and in boys between fourteen and eighteen years.

It is probably caused by some endocrine dysfunction and local trauma may play a rôle.

Pathologically it is a pericanalicular and periacinous infiltration with connective tissue and lymphocytes, together with epithelial hyperplasia.

The condition is benign and disappears within a few months.

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SIGNIFICANCE OF THE DIMINUTION OF THE SPINAL FLUID SUGAR IN TUBERCULOUS MENINGITIS

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THE present paper is a report of studies carried out to determine the diagnostic value of quantitative sugar determinations of the spinal fluid in cases of tuberculous meningitis. At the same time an attempt was made to ascertain the possible mechanism influencing the reduction in sugar content.

In the course of our studies more than one hundred cases of tuberculous meningitis were observed. The diagnosis in most cases was based on clinical signs, bacteriologic examination, as well as autopsy examination. The method used for the examination of the spinal fluid sugar was that of Jensen and Hagedorn. It should be stated that, according

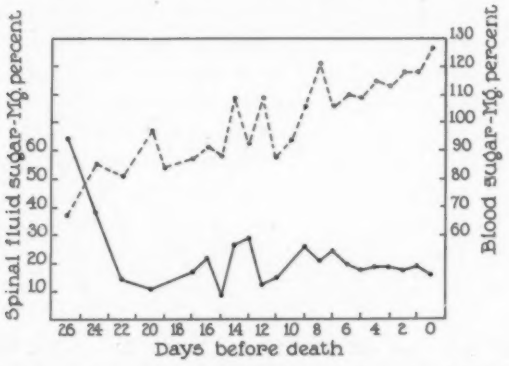

Fig. 1.—Blood sugar and spinal fluid sugar in tuberculous meningitis: 130 tests in 80 children.

to this method, the amount of sugar in the cerebrospinal fluid of fasting infants and children ranges from 35 to 60 mg. per cent. It has been shown that the reducing substance in the cerebrospinal fluid consists almost exclusively of dextrose.[1, 2] It is important, in all cases in which a quantitative estimation of the spinal fluid sugar is being made, to precede the spinal puncture by an examination of the blood sugar level. Spinal fluid drainage in itself is sufficient to cause a rise in the level of blood sugar.[3]

Figure 1 represents graphically blood sugar and spinal fluid sugar determinations of eighty children suffering from tuberculous meningitis.

The curve shows a progressive diminution of the spinal fluid sugar from the time of first observations to the fatal terminations of the cases.

From the Children's Hospital of the University of Leipzig.

In this decline there is, in many cases, a slight increase about two weeks before death, and then a final drop of the sugar content. This peculiar curve seems to be very characteristic in tuberculous meningitis although it was not observed in every patient.

We feel that the diminution of the spinal fluid sugar is an early diagnostic sign since in several cases the value of the sugar showed a decline twenty-five to thirty days before the death of the patient. In most cases the decrease in the sugar content follows a rise in globulin and an increased cell count.

Figure 1 shows, not only the gradual diminution of the amount of sugar in the spinal fluid, but also a progressive and concomitant increase of the blood sugar. This occurred in most of the single cases in which repeated examinations were made. This is surprising, in view of the fact that most of the patients suffered from malnutrition.

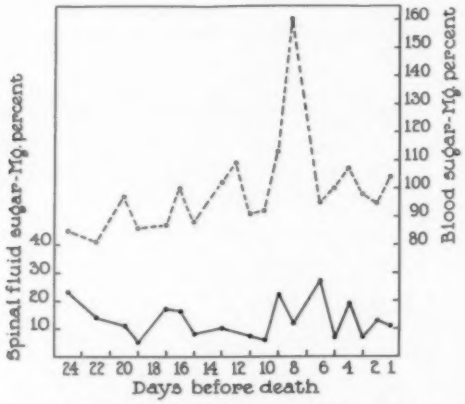

Fig. 2.—Blood sugar and spinal fluid sugar in a child suffering from tuberculous meningitis.

In observing single cases we encountered the difficulty that most patients did not come under observation until three weeks before death, so that in the average case not more than four to six examinations of the spinal fluid could be made. A few patients were observed from the onset of the disease, some with other tuberculous lesions and others free from tuberculosis when admitted to the hospital. The quantitative blood and spinal fluid sugar determinations of one of these children is shown in Fig. 2, demonstrating the diminished spinal fluid sugar level as it occurred twenty-four days before death.

In one patient, tuberculous meningitis developed following tuberculous spondylitis. When the first meningeal signs were observed, the spinal fluid revealed tubercle bacilli, but a normal sugar content, 59 mg. per cent (blood sugar, 94 mg. per cent), and only a slightly increased cell count and protein content. Five weeks later (five weeks before

death) the spinal fluid sugar dropped to 27 mg. per cent, when the first signs of meningeal irritation were observed in the spinal fluid, as revealed by increased cell counts and strongly positive protein reactions. The clinical picture in this case was always doubtful as to the presence of a basilar meningitis, since the patient was suffering from Pott's disease, with spasms of the lower extremities.

In another exceptional case the disease lasted eighteen weeks. One hundred three days before the death of the child, the spinal fluid sugar dropped to 19 mg. per cent. This value remained approximately on the same level until the fortieth day before death. Four days later the sugar was 8 mg. per cent, rising to 24 mg. per cent in the fifteenth day before death and finally diminishing again. The autopsy in this case, in contrast to the usual finding, revealed a definite productive leptomeningitis.

The question arises whether a low spinal fluid sugar in itself in cases of tuberculous meningitis would indicate invariably a fatal prognosis. As we have not observed any recoveries from this condition, we cannot definitely state this to be a fact. We may, however, reason from cases with purulent meningitis in which we have observed the return of the spinal fluid sugar level to a normal value with recovery.

In syphilitic meningitis of infants, in whom the values of spinal fluid glucose in some cases show a considerable diminution, a progressive increase in the sugar content accompanied the clinical improvement.

From a therapeutic standpoint it seemed of interest to attempt to raise the lowered spinal fluid sugar in cases of tuberculous meningitis. We injected glucose into the spinal canal in order to observe whether such injections would perhaps maintain the spinal fluid sugar at higher levels. Repeated examinations of the spinal fluid in children who were so treated showed that on the day following the injection the spinal fluid sugar had returned to the same low level as previously. Since no intraspinal glucose injections have been carried out on normal children, we have no means of determining whether a similar decrease ensues.

However, the injection of 5 per cent glucose intraspinally in children suffering from tuberculous meningitis did produce another result. We observed that there was a response in blood sugar following such injections, which can be compared with that found after intramuscular injection or after absorption of sugar from the intestinal tract.

Table I shows the rise and fall of the blood sugar after the intraspinal injection of 5 per cent glucose (30 c.c.) in a patient with tuberculous meningitis. An equal amount of fluid had been withdrawn before the injection. This may perhaps indicate that in cases of severe tuberculous meningitis the sugar center in the brain responds to the intraspinal injection of glucose with an initial intense increase and a subsequent slow decrease of the blood sugar. Regardless of whether this

change in blood sugar is a specific sequela of the sugar injection or, what is more likely, is due to the intraspinal procedure itself, it would seem that the blood sugar center in the brain can easily be influenced.

TABLE I

RESPONSE IN BLOOD SUGAR FOLLOWING THE INTRASPINAL INJECTION OF 5 PER CENT GLUCOSE

	BEFORE INJ.	½ HR. AFTER INJ.	1 HR.	1¼ HR.	2 HR.	3 HR.
Blood sugar in mg. per cent	114	201	220	222	194	148

The rapid increase of the blood sugar following intraspinal injection of glucose would tend to indicate that in tuberculous meningitis, as well as in normal individuals, substances brought into the subarachnoid space are likely to enter the brain tissue itself, or to come into such close contact as to produce an irritative effect. The presumption is that the pressure within the subarachnoid space is high enough to allow a substance to pass into the perivascular spaces of Virchow-Robin, as well as into the directly communicating perineuronal spaces.

The question arises as to whether the low content of the sugar in the spinal fluid can be explained by actual splitting up of sugar within the ventricular system or spinal canal, either by the cells themselves or by the disease-producing organisms. This has been shown to be the cause for the decrease of sugar in cases of purulent meningitis, in which both cells and bacteria are able to break down glucose. Experiments of Mader[4] have shown that a considerable decline of the spinal fluid glucose occurs after the addition of bacteria.

An indirect proof of the breaking down of glucose in the spinal fluid has been demonstrated by some investigators by showing an increase of lactic acid. However, Fasold and Schmidt[5] showed that this increase in tuberculous meningitis is not high enough to explain the diminution of the spinal fluid glucose by the breakdown of sugar. The studies of Fasold and Schmidt, as well as our own, have also shown that, when suspensions of tubercle bacilli are added to normal spinal fluid, or when spinal fluid from patients with tuberculous meningitis is incubated for from one to fourteen days, there is no breaking down of glucose. It has been shown that normally the sugar content of the ventricular cerebrospinal fluid is higher than that of the subarachnoid fluid, because of the utilization of the glucose by the brain itself or its nervous elements. The irritation of the cells of the brain in tuberculous meningitis, resulting in an increased utilization of glucose, may account partially for the diminution of the sugar.

Is there another mechanism for the decrease in the sugar of the spinal fluid in cases of tuberculous meningitis? In studying this question the sugar content of the spinal fluid was examined in some cases of tuber-

culous meningitis at the beginning and at the end of a spinal puncture after removing a large amount of fluid. In other patients the examination of the spinal fluid sugar was repeated after one hour. If it is assumed that the spinal fluid is produced in the choroid plexus with normal glucose value and then is reduced in sugar concentration by the action of bacterial enzymes, a period of time greater than the duration of a spinal drainage would be necessary to yield this reduction. Therefore, the sugar concentrations of specimens removed after withdrawal of large amounts of fluid would certainly be normal or at least greater than the original value at the beginning of the drainage. Our observations, however, showed that the sugar content at the end of a spinal puncture gave almost the same value as at the beginning and that a repetition of the puncture after one hour usually gave no higher amounts. Therefore, the possibility of glycolytic enzymes having been the sole cause of the diminution seems unlikely.

If the increased splitting up of sugar is not the only cause of the diminution, we must assume that in cases of tuberculous meningitis the spinal fluid primarily contains a lower sugar content. It seems possible to explain this low content of glucose on the basis of a diminished passage of sugar through the barrier between blood and spinal fluid. Apparently in tuberculous meningitis this threshold is raised, and instead of a normal relation of blood to spinal fluid sugar of 2:1, we find a relation of 8:1, 10:1, or even 12:1. Walter[6] states that especially in inflammatory processes of the meninges the passage for certain substances, such as protein and the bromides, is increased, but that, on the contrary, the threshold for sugar is higher.

A series of experiments was made to test the assumption that in cases of tuberculous meningitis the barrier between blood and spinal fluid is disturbed with respect to glucose. We raised the level of blood sugar in normal patients and in those suffering from tuberculous meningitis by the injection of adrenalin, or by feeding glucose and cane sugar, or by the injection of glucose intravenously.

We know that a parallel rise in spinal fluid sugar of the normal individual accompanies an artificial increase of blood sugar. We also know that the relation of the blood to spinal fluid sugar is maintained in diabetes in which we find values of 100 to 200 mg. per cent in the spinal fluid corresponding to amounts of 200 to 400 mg. per cent in the blood.

Different opinions have been expressed in the literature as to when the spinal fluid sugar reaches its maximum following augmentation of the blood sugar. Table II gives a survey of our experiments on two normal cases of our series.

The results show the response of the spinal fluid sugar to an increase of blood sugar, indicating that the increase of sugar in the normal spinal fluid follows that of the blood sugar closely. The spinal fluid remains

TABLE II

RESPONSE OF BLOOD SUGAR AND SPINAL FLUID SUGAR FOLLOWING THE ORAL ADMINISTRATION OF GLUCOSE IN NORMAL CHILDREN

		SPINAL FLUID SUGAR IN MG. PER CENT	BLOOD SUGAR IN MG. PER CENT	SUGAR ORAL
Case 1	Before feeding	58	91	
	After 1 hr.	93	165	100 gm.
Case 2	Before feeding	51	99	
	After 1 hr.	144	252	50 gm.
	After 2 hr.	144	171	

high even after the blood sugar has decreased. As it was not feasible to puncture normal children more than two to three times without any clinical indication, we cannot state how long the cerebrospinal fluid sugar would remain high and how long the increase would be maintained.

Table III gives the data of a few cases of tuberculous meningitis with a diminished content of spinal fluid sugar which we attempted to raise by

TABLE III

RESPONSE OF BLOOD SUGAR AND SPINAL FLUID SUGAR FOLLOWING THE ORAL AND INTRAVENOUS ADMINISTRATION OF GLUCOSE IN CHILDREN WITH TUBERCULOUS MENINGITIS

CASE		SPINAL FLUID SUGAR IN MG. PER CENT	BLOOD SUGAR IN MG. PER CENT	GLUCOSE
1	Before feeding	14	116	50 gm. oral
	After 1 hr.	36	256	
2	Before feeding	17	142	50 gm. oral
	After 1 hr.	35	211	
	After 1½ hr.	49	146	
	After 2 hr.	39	133	
3	Before feeding	7	100	50 gm. oral
	After 1 hr.	14	220	
4	Before injection	8	109	20 c.c. 25%
	After 10 min.	11	300	intraven.
5	Before injection	8	118	20 c.c. 25%
	After 30 min.	10	140	intraven.
	After 1½ hr.	9	105	

giving dextrose by mouth or by intravenous injection. In Case 1, after the ingestion of 50 gm. of glucose, the spinal fluid sugar showed an increase in the same proportion as the blood sugar. The percentage of increase as compared with the rise in blood sugar seems to be normal, or at least not less than that of normal individuals who show an increase in blood sugar. The absolute augmentation (7 mg. per cent in Case 3), however, is very low. Similar results were obtained after the injection of adrenalin. Thus we see that the absolute increase of spinal fluid sugar is in proportion to the original amount of sugar. Case 2 shows that the maximum of the rise in the spinal fluid sugar follows that of the blood sugar, but it is interesting to note that after one hour the value was doubled. Two hours after the feeding the spinal fluid sugar

began to fall. The augmentation of the blood sugar content, therefore, does not help much in maintaining a higher spinal fluid sugar.

These observations do not favor the theory of increased destruction of glucose, because, if we assume the decrease in spinal fluid sugar to be the consequence of glycolysis by tubercle bacilli, we would not expect such a rapid diminution within one hour after the passage of sugar into the spinal fluid. On the other hand, a careful study of the above tables shows that the original ratio between blood and spinal fluid sugar remains approximately the same. In other words, if we increase the blood sugar to double the amount, the spinal fluid sugar rises in the same proportion.

Since the mechanism of the production of the normal spinal fluid has not definitely been established, it is difficult to draw a conclusion from the results in pathologic conditions. The question has not been decided whether the spinal fluid is to be considered a dialysate or an ultrafiltrate alone or whether it is produced by active work of the cells. The results of our experiments in cases of tuberculous meningitis could hardly be explained by ultrafiltration of sugar. Evidently, in such advanced conditions as cited, the barrier between blood and spinal fluid is not entirely closed, but partially blocked so as to allow only a small percentage of sugar to pass, at least less than the normal amount of 50 per cent. If we increase the amount of glucose in the blood, the barrier allows more sugar to pass, but an amount much smaller than normal. We feel that an active secretion by the cells within this barrier is to be held responsible for this phenomenon.

Another possible explanation is that the cells within the membranes representing the barrier and regulating the passage of sugar are so definitely disturbed in tuberculous meningitis that only a small percentage of sugar can enter the spinal canal but that after a considerable increase of the blood sugar a local production or secretion of sugar follows.

Experiments have shown that the increased passage of one substance from the blood into the spinal fluid does not necessarily prove that the passage for all substances is enhanced. The passage of one of these substances, which may be crystalloids, colloids, or nonelectrolytes in molecular dispersion, such as sugar or urea, is independent of the others. According to Walter,[6] the barrier may allow crystalloids and nonelectrolytes in molecular dispersion to pass without increased passage of proteins. On the other hand, in some pathologic conditions of the central nervous system, in which the protein passes in larger amounts, the permeability for crystalloids is regularly increased. This tends to prove that in all probability specific functions of the cells regulate the passage of the sugar and that pathologic conditions affecting these functions can produce definite disturbances of the barrier leading to a primarily decreased sugar content of the spinal fluid.

SUMMARY

1. The spinal fluid sugar in more than one hundred children with tuberculous meningitis revealed a progressive diminution from the time of the first observations to their deaths. In this decline there was in many cases a slight increase a few days before death, followed by a final decline.

2. Most of these children showed a marked progressive increase of the blood sugar.

3. Following the intraspinal injection of glucose in children with tuberculous meningitis, a response in blood sugar was observed, which can be compared with that found after intramuscular injection or after absorption of sugar from the intestinal tract.

4. The sugar content of the cerebrospinal fluid at the end of a spinal puncture gave almost the same value as at the beginning, and a repetition of the puncture after one hour usually gave no higher amounts.

5. After an artificial increase of the blood sugar, the ratio between blood and spinal fluid sugar remained the same, the absolute augmentation of the glucose in the cerebrospinal fluid being very low.

6. These experiments tend to show that the chief cause of the diminution of sugar is not an increased glycolysis, but rather a disturbance of the barrier between blood and spinal fluid.

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TWISTED OVARIAN CYSTS IN CHILDREN

Report of a Case in a Ten-Year-Old Child

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OVARIAN tumors in childhood are relatively uncommon. The literature contains a moderate number of references to this condition. The seriousness of the condition when present and, more particularly, its early recognition and surgical treatment are of sufficient importance to report a case.

J. S., a white girl, ten years of age, was admitted to the Edgewater Hospital, Jan. 15, 1936, because of cramplike pains in the abdomen. Her past history was essentially negative except for a similar attack of pain experienced for the first time on Dec. 15, 1935. At that time she was awakened at night by the pain in the lower left side of her abdomen. There were no other associated complaints. An enema administered by her mother produced relief from pain in about an hour, and the child returned to school the next day.

On December 17 and December 30, 1935, she again had similar attacks of pain. The latter were associated with vomiting of undigested food, and the pains were more severe and persistent. However, the administration of enemas again produced the same relief as was effected on previous occasions, with complete absence of pain in the interim. After the last attack, medical consultation was sought, and a diagnosis of spastic colitis was made. A bland diet and medication were prescribed. After freedom from the attacks for the next two weeks, the patient was suddenly seized with severe abdominal cramps again and brought to the hospital where she was seen by us for the first time.

It seemed that the child had departed from the prescribed diet for the past twenty-four hours and the repeated vomiting attacks, both at home and at the hospital, revealed undigested foods. The administration of an enema by the mother failed to effect the usual relief from pain during this attack.

Physical Examination.—The child cried almost continuously because of pain in the lower left side of her abdomen. At times she was entirely free from pain for short intervals. She was well nourished and did not appear acutely ill or toxic. The temperature on admission was 98.6° F.; pulse, 88; and respirations, 22. Regional examination was essentially negative.

The abdomen was soft, no masses were palpable, and the only essential finding was definite tenderness to palpation in the lower left quadrant.

The blood, on admission, showed 8,850 leucocytes, 62 per cent polymorphonuclears, and 38 per cent small and large lymphocytes. The urine was entirely normal. The condition was considered nonsurgical for the time being, and the patient was placed in semi-Fowler's position, and an ice bag was applied to the abdomen. Sedatives were administered for the pain without much effect.

Course of Illness.—The patient continued to be afebrile the next day, and the pulse ranged between 88 and 108. Repeated blood examination revealed a rise in leucocytes to 14,000 with 92 per cent polymorphonuclears. She did not appear toxic or acutely ill. Pain and vomiting of undigested food continued, and enemas produced no results. Rectal examination at this time revealed a somewhat firm and tender mass the size of a small orange in the culdesac. A flat x-ray film of the

abdomen taken on admission visualized a mass which was interpreted by the roentgenologist to be a full urinary bladder. Evidences of intestinal obstruction or urolithiasis were not visualized (Fig. 1).

The possibility of ovarian tumor was now considered, but in view of all findings it was deemed better to observe the patient further since the abdominal findings and her general condition had remained unchanged.

The following morning the whole clinical picture had abruptly changed. The patient appeared acutely ill. The pain had increased in severity and was continuous. Vomiting of clear fluid continued. The legs were drawn up toward the abdomen. There were signs of mild toxicity, but still the patient remained afebrile while the pulse ranged between 110 and 128. The abdomen was tender throughout, but more so over the pubic region and both lower quadrants, where there was definite muscular resistance to palpation. No mass could be palpated. Rectal examination at

Fig. 1.

this time revealed the previously felt mass now markedly increased in size, excruciatingly tender to palpation, and of a semifluctuant consistency. A definite diagnosis of twisted ovarian cyst was now made, and surgical procedure instituted.

Treatment.—The abdomen was opened in the midline, and at once we were confronted with a dark, bloody fluid within the peritoneal cavity. On the left side was a large dark mass which appeared almost gangrenous and which had become so engaged in the pelvis that we had considerable difficulty in disengaging it from the pelvic cavity. Grossly, the mass was dark red, hard, and had cystlike areas. It was noted that this mass had become twisted on itself and had five definite twists around the pedicle, which accurately coincided with each attack of pain. The mass was removed easily by means of two clamps at its base and ligated.

Examination of the opposite tube and ovary showed them to be small and normal in appearance. The appendix was removed at the same time. The abdomen was closed in the usual manner.

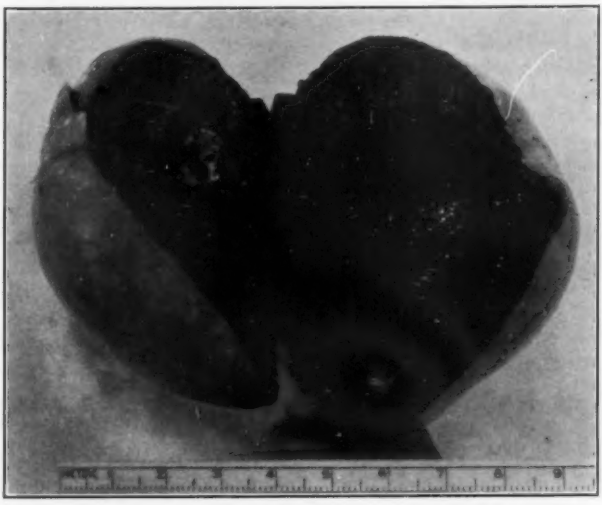

Fig. 2.

Fig. 3.

Pathologic Report.—Gross Examination: The mass was 7 by 5 by 5 cm. and globular in shape (Fig. 2). The surface was smooth and deep purple in color. A short twisted pedicle was attached. Section revealed a smooth-walled sac 5 cm. in diameter filled with serosanguineous fluid. The portion of the wall adjacent to the pedicle was thickened to 15 mm. in diameter, the remainder being thin and comprising the wall of the sac. Portions of the thickened wall contained small sacs filled with cheesy white material and hair.

Microscopic Examination: Section revealed a fibrous tissue wall so densely infiltrated with blood elements as to obscure almost completely the cellular structure. The thickened portion of the wall has a similar dense extravasation of blood with areas revealing squamous epithelium with cornification and a few typical hair follicles and sebaceous glands (Fig. 3).

Diagnosis.—Dermoid cyst with recent hemorrhage from torsion.

The child made an uneventful recovery and left the hospital on the tenth day. She has since been entirely free from pain and symptoms, except for a slight menstrual flow which appeared for one day.

COMMENT

Previous to 1905 about sixty cases of various tumors of the ovary in children had been reported, and since that time another thirty-eight have been added.[1] It is of importance to note that 60 per cent of these tumors were considered benign until 1912, but with improved histologic study it has been shown since that 60 per cent of them are malignant.[2] This finding is of importance in the consideration of the subject. The greatest number of tumors of the ovary in children occur between the ages of ten and fourteen years. Their earlier occurrence may be associated with signs of sexual precocity. The earliest reported case was that of Doran in a seven-month premature living infant.[3]

The history and clinical course of the case reported here, until marked torsion of the pedicle of the ovary took place, could easily have been attributed to a number of conditions which occur in childhood. Certainly ovarian tumor would be the least likely. Indigestion associated with dietary indiscretions, spastic colitis, left-sided appendicitis, appendiceal abscess, and the less likely conditions such as urolithiasis, and incomplete intestinal obstruction, intussusception, mesenteric adenitis were to be considered. The roentgenologic report offered no aid, although the fact was immediately appreciated after surgery that had the urinary bladder been emptied before the flat plate of the abdomen was taken, the evidence of the ovarian mass could have been suspected by visualization. This point is of great importance. In the face of the nonsurgical clinical course of our patient, the one great aid to the correct differential diagnosis lay in the rectal examination which, together with a correct x-ray interpretation, might have made possible an earlier recognition of the condition encountered here. The absence of toxicity, of abdominal surgical findings, and of fever justified medical observation with a view toward evaluating the mass palpated

rectally. Twisting of the pedicle within the following twelve hours produced an increase in the size and tenderness of the mass, with symptoms of toxicity of the patient, which clarified and substantiated the correct diagnosis, thus leading to immediate surgical intervention.

CONCLUSIONS

1. Repeated attacks of abdominal pain in children justify a complete and careful study comparable to that afforded adults. It is wrong to assume that such attacks are probably insignificant, or to ascribe them to the large mass of indefinite conditions headed by the much abused term "colitis."

2. Rectal examination done carefully and repeatedly is of utmost importance in all children with abdominal pain. This statement cannot be repeated too often.

3. The roentgenologist may be of definite assistance in the visualization of the ovarian mass quite early in the course of its development if a series of flat plates of the abdomen are taken before and after the urinary bladder is emptied.

4. Surgical treatment should be instituted as soon as the diagnosis is made, particularly because of the high frequency of malignancy encountered in tumors of the ovary in childhood.

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SPONTANEOUS RUPTURE OF THE LIVER IN A CHILD WITH COMPLETE RECOVERY

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TRAUMATIC rupture of the liver is comparatively rare, but spontaneous or hemorrhagic rupture is even more rare. There is no doubt regarding its occurrence in certain cases, some patients having been confined to bed under close observation for some time previous to the rupture. These spontaneous ruptures may be differentiated from traumatic ruptures by the fact that they are intracapsular. Anatomically the rupture is due to a hemorrhage, which may be secondary to old lesions of the hepatic parenchyma, such as secondary tumors, syphilitic lesions, or lesions of obliterating arteritis, which have been demonstrated histologically in some of these cases. According to Devic and Beriel,[2] spontaneous rupture is due to lesions of arteritis. This is an infarct at first, followed by increased vascularization at the periphery and rupture of a vessel causing an intrahepatic hemorrhage. This is followed by rupture of the glandular tissue with the production of a subcapsular hematoma. There may result a degeneration of the parenchyma and connective tissue, particularly in cases of central rupture, which are, of course, difficult to diagnose, with the formation of secondary abscesses. The classical symptoms and signs of spontaneous rupture are as follows:

Sharp pain in the right hypochondrium
Vomiting
Pallor
Anemia
Slow pulse early with rapid pulse as peritoneal irritation sets in
Jaundice
Leucocytosis
Cullen's sign of bluish discoloration at the umbilicus
Shock
Right hypochondriac mass—tense, tender, resilient
Tympanites
Rigidity
Blurring of the pleural sinus
Negative gastric, renal, and pulmonary signs.

Death may occur in a few minutes, an hour or two, a few days, or occasionally after a week or more. So far as we have been able to determine, no patient with true spontaneous rupture of the liver has ever recovered.

Mast and Streamer[1] reported a case of spontaneous rupture of primary carcinoma of the liver, which was probably brought on from a jar while the patient was driving a tractor. Devic and Beriel[2] reported

eleven cases gleaned from the literature; three of these cases were definitely due to syphilis, as no doubt were some of the others; one was due to cancer; three were uncertain or incomplete; and in four there was no history of injury, and no definite cause for the rupture was discernible. Since we have been unable to get a copy of their original article, it is impossible for us to determine whether or not some of the cases which they discussed are duplicated in the cases which we have been able to locate in the literature. Nauwerck[3] reported two cases of rupture of the liver due to stasis of bile. Genell[4] reviewed three of the four known cases of rupture of the liver in the newborn and added three cases of his own. He suggested that two accidentally coinciding lesions, both of which are individually rare, that is, a mechanical traumatic factor, contracted or distorted pelvis, plus an abnormal tendency to bleed, pseudohemophilia of the newborn, are responsible for the ruptures in these cases. Steiger,[5] Andersson,[6] Furtwaengler,[7] and Aldridge[8] each reported a case of rupture of the liver due to trauma. Watzold[9] reported a case due to syphilitic aneurysm, and Carnable[10] reported a case of spontaneous rupture of the liver in a twenty-six-year-old male, in which the direct cause was obscure. Ecarius[11] reports three cases: one due to trauma, one due to adenoma, and one due to an aneurysm.

With the exception of the cases of rupture of the liver in the newborn, which were all due to trauma, the few cases recorded in the literature were all adults; moreover only four of these were not due to trauma, syphilis, carcinoma, or adenoma; and recovery in a case of spontaneous rupture of the liver has so far as we can ascertain not been reported. We present a case of spontaneous rupture of the liver in a three-year-old child, who recovered and has remained in good health for more than two years.

Family History.—The mother, aged thirty-four years, father, aged sixty years, one half sister, aged forty-two years, and two half brothers, aged thirty-four and forty years, respectively, were all living and well. The family history was negative, so far as the immediate family knew, for syphilis or blood dyscrasias.

Patient's History.—M. A. G., a female child, three years of age, was born normally at full term. She was fed goat's milk modified with dextrimaltose until she was nine months of age. Then she was fed table food. At nine months of age she had a mild attack of measles without complications. She had had no illness since; no colds, coughs or sore throat, and no constipation. She had never had an intestinal disturbance of any kind until Feb. 8, 1934.

That morning the child was up at the usual hour and played about the room a few minutes before breakfast. Shortly after beginning the morning meal, she became ill and vomited immediately, before being taken away from the breakfast table. She was placed in bed and appeared exhausted. During the morning, she developed a temperature of 103° F. and was exceedingly pale. She was given castor oil and chicken broth; and orange juice and water were given freely. Her temperature was not taken at regular intervals until February 11, when the parents became alarmed at her extreme pallor, marked distention of the abdomen, and restlessness.

A physician was called, and she was given pituitrin, hot turpentine stupes, and enemas without relief of tympanites. At that time her temperature was 103.6° F.;

pulse, 152; and respiration, 36. For the next three days her temperature ranged from 102° F. in the morning to 104° F. about the middle of the afternoon.

When one of us saw the patient for the first time, six days after the onset, at 11 P.M., Feb. 14, 1934, she presented the following appearance.

A well-nourished, three-year-old female was resting quietly. Respiration was regular and not rapid. The head was negative; facies, anxious; skin, very pale; ears, waxy. The mucous membranes of the mouth, lips, and conjunctiva were very pale. The sclera was blue white; the pupils were equal and reacted to light. The chest was well developed; expansion was equal; and respiration was regular. Lungs were normal to auscultation and percussion. *Heart:* Apex beat was heard in the fourth interspace, 1 cm. inside the nipple line; a soft blowing systolic murmur was heard loudest just to the left of the sternum, and in the second, third, and fourth interspaces. The murmur was considered functional. The heart sounds were muffled and were not of good quality. *Abdomen:* There was a good abdominal wall, with generalized tenderness, most marked in the upper right quadrant. The liver was palpable four fingerbreadths below the costal margin and to the left of the midline. The liver's edge was rounded, soft, and tender on slight pressure. No other palpable masses in the abdomen. There was a bluish discoloration around the umbilicus. The genitals and extremities were negative.

The patient was hospitalized the following day. *Progress:* As Table I shows, her hemoglobin level at that time was 20 per cent (Dare); the red blood cells numbered 1,800,000, and the white cells, 29,150. The leucocytosis was due to an increase in young forms of polymorphonuclear leucocytes. At 11:35 A.M., a transfusion of 250 c.c. of whole blood was given by the syringe method.

Radiologic examination of the abdomen, Feb. 15, 1934, showed the liver shadow to be very much enlarged, mainly from the lower aspect, probably due to liver abscess. The abdomen was otherwise negative.

Nourishing food, plenty of fluids, medication to produce rest, and temperature sponges were given the next two days. A stool examination on the seventeenth was normal with the exception of a slight increase in mucus. Urine examinations on February 16 and 17 were normal. The patient's pulse was more feeble on February 17, but she was less toxic, and the mass had not enlarged. A blood count showed some improvement, although the hemoglobin was only 30 per cent.

On February 18, a transfusion of 210 c.c. of whole blood was given. The mass in the upper right quadrant was softer, more rounded, and extended 1 cm. lower than it had before. The left chest was more resonant to percussion, and edema of the lid of the right eye was present.

On February 20 the hemoglobin showed some improvement, and there was an even greater elevation in the white and polymorphonuclear counts; however Cooke-Ponder's index was slightly lower. There was a macular rash over the extremities, neck, and back. The temperature averages were lower, and the patient looked less toxic. There was no change in the mass.

On February 21, the patient's sclera had developed a yellowish tint, but she looked less toxic. The mass was rounded and not so tense. Neoskiodan was given intravenously, and the report of the pyelography was as follows:

On February 21, intravenous pyelography after injection of neo-iopax showed incomplete filling of the calyces of both the right and left, but good filling of both pelves. The entire right kidney was well outlined, the left not so well. The ureters were not filled well enough to make any further statement of their condition. The bladder was apparently normal.

The blood count showed improvement in that the hemoglobin was 55 per cent.

On February 22 a transfusion of 250 c.c. of whole blood was given by the syringe method.

On February 23 a hypodermatoclysis of 500 c.c. of normal saline was given.

TABLE I

BLOOD

Date	2/15/34		2/17/34		2/20/34	2/21/34		3/5/34
Red blood cells	1,800,000							
Hemoglobin	20%		30%		40%	55%		60%
White blood cells	29,150		20,000		21,200	17,200		
Coagulation time	9 minutes		6 minutes		8 minutes			
Differential								
Neutrophiles	71%		72%		82%	87%		
Arneth and Shilling: Myelocytes	1%		0		3%	1%		
Arneth and Shilling: Juvenile	1%		3%		7%	1%		
Arneth and Shilling: Band	42%		47%		38%	37%		
Arneth and Shilling: Segmenters	27%		22%		34%	48%		
Arneth and Shilling: 2 segmenters	19%		20%		29%	35%		
Arneth and Shilling: 3 segmenters	6		2%		5%	11%		
Arneth and Shilling: 4 segmenters	2		0		0	2		
Arneth and Shilling: 5 segmenters	0		0		0	0		
Lymphocytes	29%		24%		15%	11%		
Mononuclears	0		4%		1%	2%		
Eosinophiles	0		0		2%	0		
Basophiles	0		0		0	0		
Platelets								
Malaria								
Cooke-Ponder Index	1.52		1.33		1.47	1.69		
Polychromatophilia	slight		moderate		slight	slight		
Normoblasts	few		occasional		occasional			
Basophilic degeneration	occasional							
Karyorrhexia	slight							
Pyknosis						moderate		
Deliquescence			marked					
		2/15/34 Transfusion 250 c.c. Whole Blood		2/18/34 Transfusion 210 c.c. Whole Blood			2/22/34 Transfusion 250 c.c. Whole Blood	

Blood Culture: Taken Feb. 16, 1934, negative on February 17, 19, 21, 26, March 12, 17, and 30.

Urinalysis: February 16, normal; February 17, normal; February 21, normal with exception of positive acetone and diacetic acid.

Feces Analysis: Feb. 17, 1934.

Amount: 10 gm.
Character: soft
Odor: pungent
Mucus: moderate excess
Blood (occult): very slight trace
Ova and parasites: none found

Bacteria: gram-positive and gram-negative bacilli

The sclera continued to have a yellowish tint. The entire body presented a peculiar and striking appearance, that is, a color which was midway between the appearance of the skin in hemolytic jaundice and in biliary jaundice—a most peculiar and definite coppery shade.

Radiologic examination of the chest showed an absence of a line of demarcation between the liver and lung, generalized increase in density over the entire right field due to pleural fluid and compression of the lung.

On February 24 laparotomy was performed and a hematoma of the right lobe of the liver was removed and packed as follows. A high right rectus incision was made and the peritoneal cavity opened. Immediately below the incision the enlarged liver presented, with a bluish mass beneath the thin capsule, which was covered with a thin layer of fibrin. The capsule was ruptured, and the liver, which seemed to have ruptured previously, now inclosed a large clotted mass. As much as could be safely removed was taken out, leaving a transverse irregular crater in the right lobe of the liver. The opening in the liver was packed with two gauze packs, the ends of which were brought out through the incision, which was closed in layers in the usual manner.

On February 26, although the temperature was lower than it had been in the past twenty-four hours, there was an occasional râle in the upper left lung, but no bronchophony. The general condition was good.

On February 28 a small area of bronchophony was found in the base of the right lung posteriorly. There was slight dullness over the entire right half of the chest. The yellowish cast of the sclera was clearing, and the skin was less bronzed. The roentgenologist reported that radiologic examination of the chest showed the entire right side exhibiting a density due to compressed lung, surrounded peripherally by fluid. The diaphragm shadow was completely obliterated. The heart and the mediastinum were not displaced to the left.

On March 1, bronchophony from the sixth interspace to the base of the right posteriorly and bulging of the intercostal spaces from the sixth rib downward with a beginning cellulitis, were noted. The apex beat was heard just to the left of the nipple, fifth interspace. Expansion of the right lung much less than the left. Respiration was rapid.

On March 2 bronchophony was more pronounced and over a greater area. Percussion note was not so flat, and there were no râles.

On March 3 the area of dullness had increased. There was less bronchophony, and no râles. Apex beat just to the right of the nipple line.

On March 6, 180 c.c. of fluid aspirated from the right chest.

Convalescence from the operation of the liver was more or less uneventful. The removal of the packs was accomplished after thirteen days, with some trepidation and difficulty, but no untoward results. On March 8, there was no increase in dullness since aspiration. Apex beat was in normal position.

On March 13, 1934, she was dismissed from the hospital.

CONCLUSION

A three-year-old female child, presenting a history of sudden onset of acute intestinal distress, with marked pallor and distention of the abdomen due to a mass in the upper right quadrant (which was definitely felt to be an enlarged liver), and a striking bronzing of the skin with blue discoloration around the umbilicus which led to a diagnosis of spontaneous rupture of the liver, was successfully operated upon, after the general condition of the child had been improved; she has remained in good health for over two years.

We present this case because of the rarity of the condition, the rarity of diagnosis without benefit of autopsy, and because of the gratifying outcome of our efforts, hoping that, as these rare cases are reported, sufficient data will be accumulated to reduce the difficulty in diagnosis.

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FURTHER ROENTGENOGRAPHIC STUDIES OF THE SELLA TURCICA IN ABNORMAL CHILDREN

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THE roentgenographic findings as to the sella turcica in 104 normal children were reported in May, 1921.[1] That study was undertaken as an attempt to establish, if possible, normal sellar appearance and formation as a basis for comparison with the changes in abnormal children. The conclusions reached were briefly as follows:

1. The shape of the sella turcica in normal children falls in a general way into three groups: A, circular; B, oval; and C, flat or saucer shaped, with modifications in each.

2. There is a marked variation for each age, in both the height and length of the sella for that particular age. The average height and length of the sella show comparatively rapid increase in the first two years with gradual yet irregular increase from then on up to the age of twelve years.

3. There is no relationship between the shape of the sella and the size of the head except in the case of the flat type which was always found in small heads. It does not necessarily follow that all small heads exhibit this type.

4. There is apparently no influence of sex on either the appearance or formation of the sella.

5. There is apparently no relationship between the size of the sella and the size of the head.

Our results in this series of normal children serve as controls in the series herein reported. Although the literature abounds with numerous statements as to the appearance or formation of the sella turcica in various diseases, there have been very few control studies of normal sellae.

The present report deals with four problems:

1. Does the shape of the sella turcica in abnormal children differ from that in normal children?

2. Are there any characteristic differences in the formation of the sella in various types of abnormal children?

3. Is there a difference in size of the sella in normal and abnormal children?

4. Has the appearance, formation, or size of the sella any clinical or diagnostic significance?

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From a roentgenographic viewpoint, the changes in different sellae are those of general shape, changes in the development and shape of the posterior clinoid processes and dorsum sellae, and changes in the development and shape of the anterior clinoid processes. The middle clinoids are more nearly constant.

The technic used in a study of this kind is important, not so much the exact technic used, but the constancy with which it is followed. In adults, teleroentgenography is, of course, the most exact method, but in children the long exposures necessary in this technic are not practical. We have therefore selected a tube film distance of 26 inches and have, by using a high penetration and milliamperage, been able to obtain good films with 1/8-second exposure. The tube carrier is so adjusted that the central ray passes along a line tangent to the cornea of both eyes and is then moved backward until the central ray passes through the head at a point about one-third of the distance from the external auditory meatus to the glabella. In most instances this technic gives a good projection of the sella. One can readily imagine that in some rather badly trained infants the following of any technic would be difficult even when only an 1/8 of a second immobility is required. Duplitized films and screens were used.

SHAPE OF THE SELLA

In our normal series the circular or round sella was more frequently noted. Camp,[6] who utilized our classification in a study of 1,000 sellae, mostly of adults, also found that the circular and oval predominated, but in a different ratio from ours: namely, circular, 22.5 per cent; oval, 67 per cent; flat, 15 per cent. This demonstrates that there is little difference in the occurrence of the three groups in normal and abnormal individuals. Both the circular and oval predominate and the flat shape occurs the least frequently of all. No one shape predominated in any individual disease.

TABLE I

COMPARISON BETWEEN SELLAE OF NORMAL AND ABNORMAL CHILDREN

	NO. OF CASES	SHAPE OF SELLA			PRESENCE OF SLIT		
		A ROUND	B OVAL	C FLAT	NOT PRESENT	PRESENT UNCOVERED	PRESENT COVERED
		%	%	%	%	%	%
Normal children Series I	104	66	24	10	22	39	38
Abnormal children Series II	64	43	42	15	18	33	48
Abnormal children Series III (present)	168	42	48	10	11	32	57
Abnormal children Series II, III	232	43	46	11	13	32	55

The findings in the present and previous studies on abnormal children in comparison with those of normal children are given in Table I. They confirm our previous conclusions.

FORMATION OF THE SELLA

To appreciate properly the formation of the sella turcica as depicted on an x-ray film, it is necessary to visualize the anatomic relations. The sella exists as a depression in the superior surface of the body of the sphenoid, limited anteriorly by the middle clinoid processes and posteriorly by the dorsum sellae, which carries the posterior clinoid process on either side of its tip. The middle clinoids are directly lateral and posterior to the olivary eminence. The sella is covered by an extension of the tentorium cerebelli (the diaphragma sellae) which is attached to the middle and posterior clinoid processes. All of the pituitary body with the exception of the base of the infundibulum lies below this tentorial extension. The anterior clinoid processes are formed by the medial ends of the posterior border of the lesser wings of the sphenoid; they are located at a considerable distance lateral to and above the sella turcica. The olivary eminence and the optic chiasmal groove are both extrasellar since they lie anterior to the middle clinoid processes. Differences then in configuration of the sella caused by the difference in position of the anterior clinoid processes and variations in height of the olivary eminence can mean absolutely nothing referable to the pituitary body.

In our first paper on normal children we found that "in some instances the anterior clinoids are raised above the level of the middle clinoids and are very long. In these latter cases a slit is seen running anteriorly from above the middle clinoid processes under the anterior clinoids. This slit is no way a part of the sella."

This excavation is made up below of the optic chiasmal groove anteriorly and the olivary eminence posteriorly, above by the posterior por-

TABLE II

COMPARISON BETWEEN SELLAE OF ABNORMAL CHILDREN

DISEASE	NO.	SHAPE OF SELLA			PRESENCE OF SLIT		
		A CIRCULAR %	B OVAL %	C FLAT %	NOT PRESENT %	PRESENT UNCOVERED %	PRESENT COVERED %
Mongolism	28	41	48	1	11	57	32
Adiposogenital dystrophy	79	44	47	9	8	30	62
Anterior pituitary deficiency	11	36	45	19	10	80	10
Mental retardation	74	41	50	9	23	29	48
Hypothyroidism	28	45	42	13	32	7	61
Hyperthyroidism	4	50	25	25	25	0	75
Hypogonadism	4	50	25	25	0	25	75

tion of the inferior surfaces of the lesser wings of the sphenoids, the tips of which are the anterior clinoid processes. We have designated the excavation as being uncovered or covered. By an uncovered excavation is meant one in which the lesser wings of the sphenoid are high enough to permit a visualization of the slit in the lateral view. In a large number of cases, both normal and abnormal, the slit is easily discernible, but it is partly covered by the lesser wings of the sphenoid—this we have termed a covered slit.

Fig. 1.—Normal children. Contour of sella turcica and uncovered slit below anterior clinoid process.

Timme in 1921[5] reported the presence of an excavation under the anterior clinoid process in 23 out of 24 mongolian imbeciles. He believed that there was some link between the appearance of the anterior process and this disease and further stated that "the excavation is seen occasionally in persons who have not mongolian characteristics but who have isolated symptoms and signs due to anterior lobe disturbances, namely, growth and genital abnormalities." This belief has not been confirmed by others who studied the subject on a large series. Clift[2] and Stevenson

and Stultz[3] state that this appearance is not characteristic of mongolianism but that it may be a corollary of the peculiar facial arch present in that disease.

Tables I and II show the incidence of each type of excavation in each individual disease studied and also in normal children. A covered or uncovered slit is noted in 87 per cent of the abnormal children and in 77 per cent of the normal. An uncovered slit is found most frequently in the following conditions in the order named: anterior pituitary de-

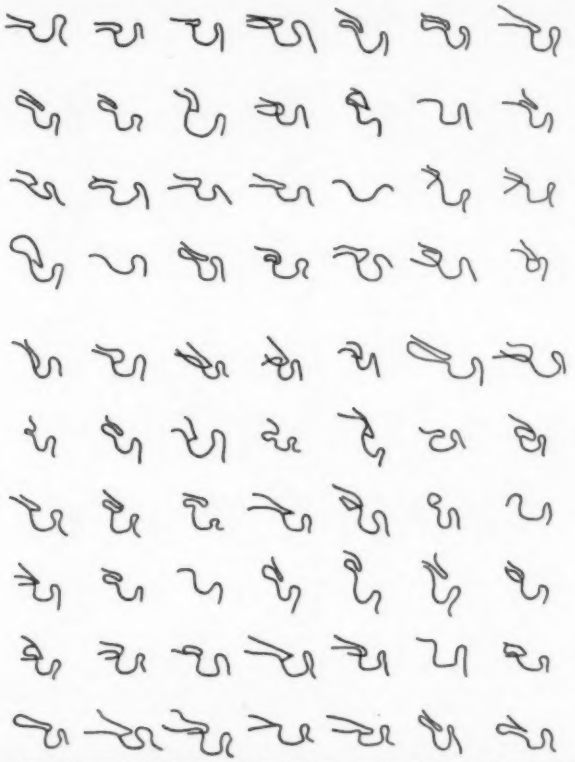

Fig. 2.—Adiposogenital dystrophy and obesity. Sella turcica shows variations in size and shape of all processes. Examples are seen of slit (*5, 6*), covered slit (*1-4*), and absent slit (*13, 19*).

ficiency, mongolian imbecility, normality, adiposogenital dystrophy, mental retardation, hypogonadism, and hypo- and hyperthyroidism. We feel that the small number of sellae studied in each disease entity prevents us from arriving at any definite conclusion as to the significance of the order of frequency. We are justified, however, in stating that its presence is not pathognomonic of any particular disease.

The shape of the slit is certainly not characteristic of any of the diseases studied as can be seen by even a casual examination of Figs. 1, 2,

3, and 4. In fact it is impossible to differentiate by means of this excavation between the sellae in the various abnormal conditions: mongolian imbecility (No. *6, 10, 11,* Fig. 3), mental retardation (No. *15, 21,* Fig. 3), childhood myxedema (No. *5, 14,* Fig. 4), hypogonadism (No. *24,* Fig. 4), lipodystrophy (No. *29,* Fig. 4), hydrocephalus (No. *40, 42,* Fig. 4), adiposogenital dystrophy (No. *5, 6, 7, 8, 34, 36,* Fig. 2). Similarly, every variation and type of the abnormal group is duplicated in the normal (Fig. 1). Comparison of the covered excavations shows that it is also impossible to differentiate between normal and abnormal (Figs. 1, 2, 3, and 4).

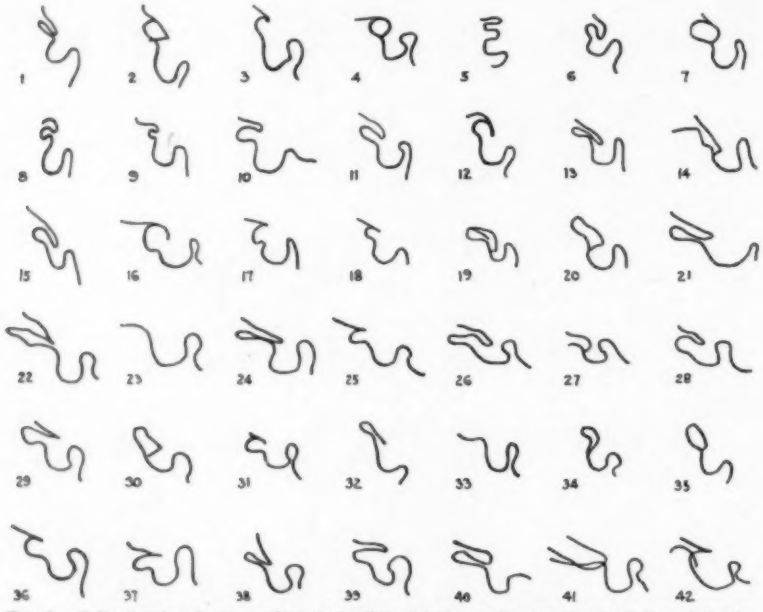

Fig. 3.—Sella turcica in mongolian imbecility (*1-14*) and mental retardation (*15-42*). Example *5* is of seven-week-old infant

The type of sella without any excavation occurs in 22 per cent of the x-ray films of normal children and in from 8 to 32 per cent of the abnormal. Here also every variation of formation and contour appearing in the abnormal children is duplicated in the normal.

The characteristics of the anterior and posterior clinoid processes are not constant in any particular disease as may be noted in studying Figs. 1, 2, 3, and 4. They show similar variations in size, shape, and development in both normal and abnormal children. The dorsum sellae, which is of more importance in this discussion than the anterior clinoid process, which is extrasellar, may be broad with rounded or bulbous posterior process, narrow with long process, or pyramidal shaped with

an apical clinoid. It may be long or short and point forward, upward, or backward. It may be well developed or poorly developed.

The dorsum sellae in adiposogenital dystrophy tends to be broad and well developed with at times a bulbous posterior clinoid process. In a few instances, the dorsum sellae and posterior clinoid may be poorly developed. The well-developed dorsum sellae is found more frequently in all of the diseases studied than the poorer developed form (Table III). In mongolian imbecility the broad dorsum sella is infrequent as compared to the pyramidal type and the long, curved narrow process.

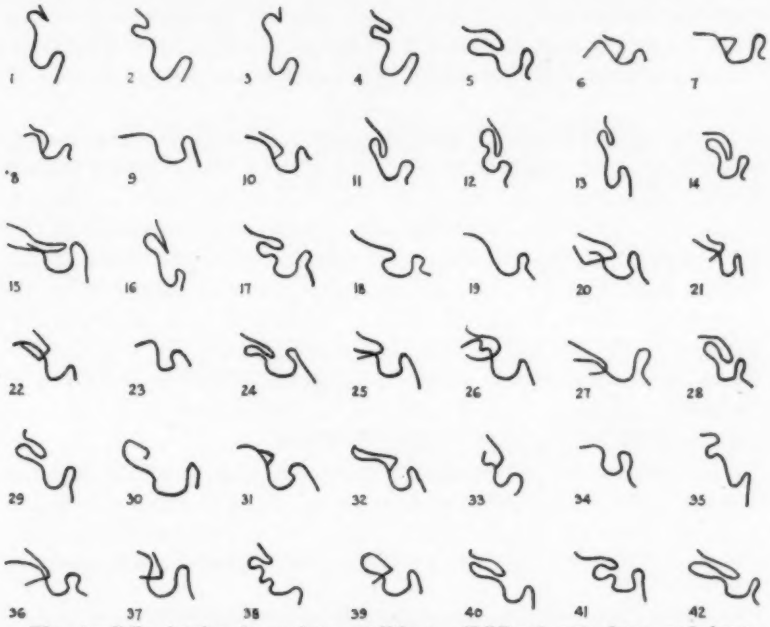

Fig. 4.—Sella turcica in various conditions. Childhood myxedema and hypothyroidism (*1-17*); anterior pituitary deficiency and infantilism (*18-23*); hypogonadism (*24-28*); lipodystrophy (*29*); achondroplasia (*30-31*); diabetes insipidus (*32*); microcephalus (*33-35*); hyperthyroidism (*36-37*); hydrocephalus (*38-42*).

Bridging and near bridging, that is approximation of the anterior and posterior clinoid processes, was encountered in only 8 instances in the series, all in the obese group.

SIZE OF THE SELLA

The dimensions of the sella turcica on x-ray films are generally expressed in terms of vertical and anteroposterior measurements. There is no uniformity, however, as to the points from which these measurements are taken. Camp[6] states that "the anteroposterior measurements are taken from the most dorsal point of the tuberculum sellae in the sagittal plane to the anterior edge of the dorsum sellae in the same

plane. The vertical dimension or height is taken along a line dropped from the anteroposterior line to the deepest point on the floor of the sella." Royster and Rodman[7] consider the height to be "a line drawn from the lower limits of the curves of the clinoid processes to the floor of the sella turcica." They do not designate the particular clinoid process, i.e., anterior or middle to the posterior, but evidently refer to the middle as their height dimensions are shorter than ours in the first series.

In our first two series[1, 4] we measured the sella as follows: the length, from the middle clinoid process to the farthest posterior part of the sella; the height, on a line joining the tips of the anterior and posterior clinoid processes, a perpendicular was drawn to the deepest portion of the sella.

In the present study we have changed our points of measurement, especially that of height since we feel that the anterior clinoid process is not a part of the sella per se and therefore should not be considered in the dimensions. In the present series the height is computed as follows: a line is drawn from the most anterior and upper point of the dorsum sellae forward to the nearest point of the dorsal aspect of the tuberculum sellae. From this line or its extension, a perpendicular dropped to the deepest part of the sella represents the height. The length is the longest anteroposterior diameter. We use Royster and Rodman's figures as normal averages for the present study as their measurements are practically the same as ours.

While our former measurements cannot be used as criteria for the present study, the general trends noted by us in the first two series, especially as to the relationships between the size of the sella and the size of the head and the increase in the size of the sella with age, are still valid.

TABLE III

APPEARANCE OF DORSUM SELLAE WITH POSTERIOR CLINOID PROCESS

	BROAD DORSUM SELLAE WITH ROUNDED POSTERIOR PROCESS			BULBOUS POSTERIOR CLINOID	POINTED POSTERIOR BROAD BASE DORSUM SELLAE	LONG CURVED NARROW POSTERIOR	POORLY DEVELOPED DORSUM SELLAE
	MEDIUM	SHORT	LONG				
Adiposogenital dystrophy	20	10	3	15	12	8	6
Anterior pituitary deficiency	3	3	0	2	2	0	1
Mental retardation	14	13	3	7	8	4	5
Mongolism	1	1	0	1	3	5	2
Hypogonadism	1	1	0	0	1	0	1
Hypothyroidism	2	3	0	3	1	0	1
Hyperthyroidism	0	0	1	1	0	0	0
	41	31	7	29	27	17	16 168

The variations in the normal series were so great that it was difficult to arrive at an average for any particular age. The same variations in size were noted in our first series of abnormal children, and it is therefore difficult to state whether they represent the normal variations. It seems that the type of disease has apparently no influence on the size of the sella per se, for the differences are not constant enough for any one disease to be of any diagnostic value. The average size of the sella turcica in various diseases is given in Table III. The sellae in the adiposogenital dystrophy group are slightly larger on the average than those in the other groups, but this increase in size is not great enough to be characteristic.

TABLE IV

AVERAGE SIZE OF SELLA TURCICA IN VARIOUS DISEASES COMPARED WITH THAT IN NORMAL CHILDREN

AGE	1-2-3 YR.	4-5-6 YR.	7-8-9 YR.	10-11-12 YR.	13-14-15 YR.	15 YR. +
*Normal**	6 × 4 mm.	7.7 × 5.4 mm.	8.4 × 6.2 mm.	9.3 × 6.0 mm.	9.6 × 6.8 mm.	mm.
Adiposogenital dystrophy		6.2 × 5.5	8.9 × 7.1	8.7 × 7.3	9.0 × 7.7	7.7 × 7.7
Mental retardation	6.8 × 6.0	8.7 × 7.0	8.3 × 8.7	8.4 × 7.4	8.0 × 6.0	
Mongolism	5.5 × 5.5	7.5 × 7.3	7.7 × 7.0			
Hypothyroidism	8.5 × 7.0	9.2 × 7.7	7.0 × 5.0 (1)		10.0 × 9.0 (1)	
Anterior pituitary deficiency		8.0 × 5.5	5.0 × 6.0 (1)	8.5 × 8.5	7.5 × 7.5	11 × 4 (17 yr.) 8 × 9 (18 yr.)
Hypogonadism			7.5 × 6.5	12.0 × 10.0 (1)	11.0 × 8.0	

*Averages taken from Royster and Rodman.[7]

One expects to find smaller sellae in children with anterior pituitary deficiency of growth, but in the present series five are below the average for the respective age, three are normal, and one is exceedingly large (10 mm. by 5 mm. in a five-year-old child).

To emphasize our statement that there is no relationship between the size of the sella and the size of the head, we note that in a microcephalic idiot ten years of age the size of the sella is 10 mm. by 10 mm., which is greater than the average for that age, while a child with hydrocephalus in the same age group has a sella which measures 8 mm. by 7 mm.

The average size of the sella in microcephalus is 7.5 mm. by 7.3 mm., irrespective of the age, while that in hydrocephalus is 8 mm. by 7 mm.

The size in nonendocrine mental retardation falls fairly within the normal limits with slight upward or downward trends in different age groups. The same tendencies are noted in hypothyroidism. The trend in mongolian imbecility is toward a smaller sella than the normal. In

two cases of achondroplasia the sellae are both much larger than the average for the respective ages.

SUMMARY

Studies were carried out on the sella turcica of 168 abnormal children suffering from adiposogenital dystrophy, obesity, anterior pituitary deficiency, hypothyroidism, mongolian imbecility, mental retardation, etc., and the findings were correlated with those of 64 abnormal children and 104 normal children previously studied. We are able to confirm our previous findings that the shape of the sella does not differ significantly in normal and abnormal children. The type of disorder is not correlated with the shape of the sella. Round and oval shapes predominate both in normal and abnormal children, and flat sellae are the least frequent.

The formation of the sellae does not differ in abnormal children from those in the normal children, nor is there any formation characteristic of any individual disease. The excavation beneath the anterior clinoid process is extrasellar and is not significant of mongolian imbecility nor of any other disorder studied in this series. The size of the sella is not influenced by any of the diseases. There is no relationship between the size of the sella and the size of the head.

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MENINGITIS DUE TO THE MORGAN BACILLUS

Report of a Case

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NUMEROUS bacteria have been reported as causative organisms in meningitis. In a review of the literature but one report of meningitis due to the Morgan bacillus has come to my attention. The following is believed to be the first case of this type of meningitis to be reported in the American literature.

The patient, a colored male, aged eleven months, was admitted to the Children's Hospital on June 13, 1935, with a history of cough and rhinitis of two weeks' duration, listlessness, and anorexia. Three days before admission he developed a discharge from his right eye and the day following he vomited once. At all times his bowels were regular, and the stools were of normal consistency.

The past history of the child was essentially negative. He was a full-term baby, having an uneventful birth and a birth weight of 6 pounds and 7 ounces. He was never ill and was given orange juice but no cod liver oil for rachitic prevention.

The physical examination on admission revealed an acutely ill colored male of about one year of age. The head examination presented a discharge from the right eye, hypertrophied tonsils, and an injected pharynx. The chest showed diminished breath sounds and a percussion note over the right base posteriorly. The heart and abdomen were essentially negative, as was the neurologic examination. The admission diagnosis was a rhinopharyngitis, catarrhal conjunctivitis, and a possible early pneumonia.

For several days the chest signs remained unchanged, and three days after admission the temperature, which was 101° F., reached 104° F. rectally. It continued to vary between 99° and 103° F., and four days after admission an x-ray plate of the chest disclosed the heart, lungs, and thymus to be normal. Smears from the eye were negative until June 15, when an organism having the morphology of the Koch-Weeks bacillus was reported.

Throughout this time the bowels had been moving only once or twice a day, and the stools were quite normal in character. The blood count gave a 44 per cent hemoglobin (Newcomer), 3,820 red blood cells, 21,000 white blood cells with 81 per cent polymorphonuclear leucocytes including 42 bands, 27 lobulated, and 2 young forms. There was a marked achromia, anisocytosis, and poikilocytosis.

On June 24 the temperature, which had slightly dropped to an average of 101° F., again rose to 104° F. At this time a bulge of the anterior fontanel was noted, and another neurologic examination was entirely negative. A spinal puncture was done, the fluid being removed under a pressure of 24 mm. of mercury, rising to 30 mm. under jugular compression with the child crying during the examination. The fluid was colorless but had a granular appearance. That evening 130 c.c. of slightly cloudy spinal fluid was withdrawn, and 30 c.c. of antimeningococcus serum was given intraspinally. Thereafter a daily spinal tap was done.

The laboratory report on the first spinal fluid showed a cell count of 250 per c.mm. with 8 per cent lymphocytes and 92 per cent polymorphonuclear leucocytes. There

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was a normal reduction of Benedict's solution, and the globulin was not increased, although the second specimen showed an abnormal amount. Examination of the culture of the first specimen of spinal fluid disclosed the finding of a gram-negative bacillus. Later the laboratory reported that this organism appeared to belong to the salmonella group and might be the *Salmonella morgani*. From the time of the first culture until the third week before discharge the cultures for this organism were consistently positive. Throughout the course of the illness there was a wide variation of the differential cell count of the spinal fluid. The lymphocytes ranged from 10 per cent to 90 per cent with a corresponding change in the polymorphonuclear leucocytes and the total count varied from 170 per c.mm. to 2,000 per c.mm. A number of blood cultures were taken and were all negative. Stool cultures were consistently negative as were several nose and throat cultures.

On July 15 the intraspinal administration of an autogenous vaccine was begun, 0.1 c.c. of the vaccine being given, the dose being increased 0.1 c.c. daily until 1.0 c.c.

Fig. 1.—Morgan bacillus meningitis. Photograph taken five weeks after admission. The spinal fluid cell count at this time was 1,700 per c.mm.

was given. The cell count rapidly rose to 1,400 per c.mm. and then the vaccine was stopped. The count receded until the last puncture was done on August 5 when it was 160 per c.mm. In general the patient looked well and was discharged August 13, two months after admission, as cured. The child was seen several times about two months later and seemed to be in the best of health.

COMMENT

As we review the case we are impressed with several striking features. One is that, in a case of meningitis with a spinal cell count mounting to 2,000 per c.mm., at no time did the child ever have any of the neurologic symptoms of meningeal infection, in fact, the child was never acutely ill. During the course of his illness he learned to stand and even walk a few steps. The picture of the patient was taken five weeks after admission, and at the time his spinal fluid cell count was 1,700 per c.mm.

Another interesting fact was that the stool cultures and the blood cultures were repeatedly negative. One can rule out infection of the spinal tract from lumbar puncture by the obtaining of the positive culture from the very first specimen of spinal fluid.

To check the diagnosis of a Morgan bacillus infection further, a specimen of the spinal fluid and one of the laboratory's cultures were sent to the National Institute of Health. Their examination showed the presence of gram-negative motile rods. The fermentation reactions were studied on twenty-five different sugars and a number of media. The organisms studied produced acid and gas in dextrose, galactose, levulose, mannose, and a trace of acid in glycerin. After seven days the following were completely negative: adonitol, amygdalin, arabinose, dulcitol, dextrin, erythritol, inositol, inulin, lactose, maltose, mannitol, raffinose, rhamnose, saccharose, salicin, sorbitol, starch, trehalose, and xylose. The methyl red reaction was positive. Gelatin was not liquefied and litmus milk was neither acidified nor coagulated. Indol was formed and lead blackened. The nitrates were reduced. Agglutination serums for the Morgan bacillus were not available but "cartrol" agglutination tests with serums for the typhoid, dysentery, and various salmonella strains were all negative. In the opinion of Dr. Branham, of the National Institute of Health, agglutination by specific serums was entirely unnecessary as the organisms had such clear-cut cultural and biochemical characteristics. The lack of maltose fermentation and positive reduction of nitrates were the only deviations from the classic strains first described by Morgan.

A review of the literature reveals only one other case of a Morgan bacillus meningitis.[1] The symptoms in this case were those of a typical meningitis ending fatally. The Morgan bacillus is primarily a habitant of the intestinal tract both normally and in the presence of a diarrhea.[2] It has often been reported as a common cause of diarrhea in infants,[3] especially in the summer time.[4] The extent of the infection of the Morgan bacillus is not limited to the intestinal tract. Magath and Jackson[5] in 1924 reported a case of a fatal septicemia due to this organism, and similar infections have also been reported by others.[1] It has been known to produce pyelitis,[6, 7] ulcerative colitis,[8] and a fatal cholecystitis[9]; d'Aunoy[10] also reports colitis and pyelitis. Through a series of thirteen cases Havens and Ridgway[11] studied the pathogenicity of the Morgan bacillus and its wide range of infection. The organism occasionally gives rise to a diarrhea similar to a paratyphoid infection[12] and the patient's serum gives a positive agglutination with the Morgan bacillus.

In general, the Morgan bacillus[13] is seen to be a gram-negative motile rod which ferments only glucose, levulose, and galactose. It causes a strong indol reaction in peptone water and will not cause any growth in uric acid or citrate media. In Jordan's citrate medium it causes an acid reaction in the butt and slant. This organism has been observed to be nonpathogenic for laboratory animals if obtained from healthy individ-

uals and fatal to them if obtained from persons ill with the disease. The Morgan bacillus has been seen not only in man, but also in cows, monkeys, mice, snakes, parrots, and even roaches, and has been obtained from the soil and water.

It can thus be seen that the distribution of the Morgan bacillus and its infectiousness is not limited to the gastrointestinal system. It has even been seen in conjunction with mastoiditis[14] and with mental disease.[15]

SUMMARY

1. A case of Morgan bacillus meningitis is presented, and it is believed to be the only case reported in the American literature.

2. There is a brief review of the extent of the infectiousness of the organism.

3. The cultural and biochemical characteristics of the Morgan bacillus of the case are presented.

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CITRATED TRANSFUSION IN INFANTS AND CHILDREN

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RECENTLY I described an automatic two-way valve for use in transfusions and other therapeutic procedures. I wish to report its successful use in over 125 transfusions since January, 1935, on the pediatric service of Mount Sinai Hospital. Here it is employed for citrated transfusions in infants and young children. Experience with the unit in these cases emphasized the ease and desirability of utilizing this automatic device. It facilitates the injection of the citrated blood into the recipient under pressure. This permits the use of a fine hypodermic needle of a 23 or 25 gauge, thus eliminating in most of the cases, the necessity of cutting down to enter the vein. In the series of cases here

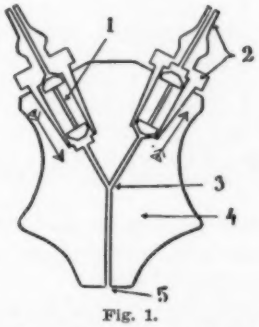

Fig. 1.

Fig. 2.

Fig. 1.—Cross-section. *1*, Automatic valve without washers or packing. *2*, Fitting with knurled nut and end tapered for needle or rubber hose. *3*, Flow of fluid in "Y" arrangement. *4*, Stainless steel body with finger grips. *5*, Inlet for adapters for either Luer or Record syringe or for Luer syringe itself.

Fig. 2.—Apparatus taken apart.

reported 3.8 per cent gave reactions (chill or fever). It is desirable that even this small incidence of reactions should be avoided if possible. The cleaning of the apparatus after transfusion is a little more difficult than the cleaning of a needle. I am experimenting in altering the cleaning technic and in changing some little details of the apparatus with the hope that the number of reactions will be reduced to a negligible figure.

The apparatus is constructed entirely of stainless steel. Its five parts, body, two valves, and two fittings, are easily taken apart, making for thorough cleaning and sterilization of the individual parts. The valves

From the pediatric service of Mount Sinai Hospital.

are interchangeable and reversible making them foolproof. The fittings are tapered for either needles or rubber tubing. The shape of the apparatus is especially adapted for easy grip with the fingertips. The attachment for the syringe permits the use of a lock type syringe, thus preventing the separation of the syringe and apparatus during the operative procedure. The device has arrows stamped on both sides of the body to indicate the direction of the flow.

Figure 1 shows a cross-section of the apparatus, and Fig. 2 shows the apparatus taken apart.

Figure 3 shows the set-up for the extremely simple method of transfusing citrated blood. The blood is first collected from the donor,

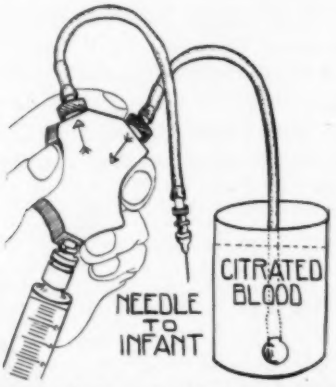

Fig. 3.—Set-up for infant transfusion using citrated blood and a small hypodermic needle with lock type syringe and the automatic two-way adapter.

using 1 c.c. of 2.5 per cent sodium citrate solution for each 10 c.c. of blood to prevent clotting. Then the needle is connected to the recipient as illustrated, and the transference of the blood begins.

In operating the instrument, one merely grips it with the fingers of the left hand in the curves especially made for convenient holding, while the syringe lies in the palm of the left hand. The upstroke of the piston automatically opens the inlet valve and closes the outlet valve filling the syringe with citrated blood from the beaker; conversely the downstroke closes the inlet valve and opens the outlet valve injecting the blood in the syringe into the recipient.

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Critical Review

NEUROLOGIC CONDITIONS IN INFANTS AND CHILDREN

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THE complications of the central nervous system that may occur during the course of some of the exanthems were reviewed[1] last year and at that time the similarity of both the gross and microscopic pathology was discussed and the various opinions as to the possible etiologic factors were considered. As the same questions are pertinent to the complications of the common contagious diseases which were not included in the previous review, it appears appropriate to consider them at this time.

Smallpox.—Complications of the central nervous system have been observed during the course of variola since the earliest recognition of the disease. Yet the frequency with which these conditions arise has been variously stated: Rolleston[2] found only 25 cases among 10,000 patients; Shamberg and Kolmer[3] observed 8 instances among 3,000; Troup and Hurst[4] report that in 51,243 cases of variola occurring in England and Wales from 1925 to September 30, 1929, there were only 140 deaths, and of this group "central nervous system complications contributory to death" appeared on the death certificates of only 25 patients. The variability in the frequency with which these complications are encountered would suggest that they occur more often during endemic or epidemic appearances of the disease than during those periods when it appears in a sporadic manner.

Clinically, the neurologic complications may vary from a peripheral neuritis (such as the case described by Putnam,[5] in which there was paralysis and subsequently atrophy of the serratus magnus alone) or hemiplegia[6] to severe and often fatal instances of encephalomyelitis.[7, 8] However, either the encephalitic or spinal types of disturbances can usually be recognized according to the predominance of the symptoms. The onset of these manifestations, which usually develop between the fifth and thirteenth day after the appearance of the rash and bear no relationship to the severity of the infection, may be sudden or may be heralded by the gradual development of somnolence, headache, unsteady gait, and alterations in behavior, or by severe pains in the abdomen, extremities, and back. In the encephalitic group the onset is usually preceded by inability to enunciate distinctly and a somnolence which rapidly passes into coma and terminates within a few hours in death. This is well exemplified in the case reported by Marsden[7] of a seventeen-year-old boy who on the seventh day of the rash complained of headache and was unconscious at the end of twenty-four hours. Death occurred forty-eight hours after the initial symptoms. However, should the patient survive the acute phase of

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the disease, recovery may be expected although the patient may remain unconscious for days or weeks. In the spinal type of involvement paralysis of the extremities constitutes the chief clinical sign. The paralysis is usually preceded by the development of stupor, severe pains in the extremities, the abdomen, or in the back, nausea, vomiting, and progressive loss of sensation and sphincter control. In the majority of instances the drowsiness or stupor disappears after a few days, but the paralysis may be present for months before recovery is made. Some of the reported instances of transverse myelitis[9, 10] would suggest a form of Landry's paralysis, such as the case of Gross[9]: A woman, fifty years of age, first developed weakness of the lower extremities, followed by involvement of the respiratory muscles, upper extremities and swallowing; death occurred at the end of seventy-two hours. The prognosis in either of the two types, i.e., spinal or encephalitic, is poor but tends to be grave in the latter group.

Unfortunately, the literature contains very little information in regard to the alterations which may occur within the *cerebrospinal fluid*. The observations of Marsden and Hurst,[11] however, would indicate that very early in the course of the disease there is a pleocytosis of either lymphocytes or polymorphonuclear leucocytes but that later the cytology is practically entirely of the lymphocytic series. Usually there is no increase in either the sugar or protein content of the fluid; bacteriologic studies have been uniformly negative.

Although the pathology of encephalomyelitis occurring in the course of certain of the exanthems has been carefully studied and is now generally recognized as possessing characteristic features, yet the etiologic factor remains debated. In fact, there appear to be two very divergent views: One group expresses the opinion that the disturbance is due to the virus of the specific disease, while the other is of the opinion that the complication is secondary to an inflammatory process initiated by the activation of a neurotrophic virus which under normal conditions remains dormant. The same controversial situation arises in the discussion of encephalomyelitis following vaccination. Thus Troup and Hurst,[12] after a careful study of the literature and a case of their own, concluded that the demyelinization, perivascular infiltration, and degeneration occurring in postvaccinal encephalitis were indistinguishable from those changes encountered in disseminated encephalomyelitis following measles or smallpox. McIntosh and Scarff[13] in 1930 concluded that the virulent strains of the virus of smallpox when injected intracerebrally, intravenously, or intradermally into rabbits produced a definite meningoencephalitis and that the lesions produced were comparable to those observed in postvaccinal and postvariola encephalitis in man. Jorge[15] in a recent review of this type of encephalitis and other allied conditions has discussed both views concerning the etiology of postvaccinal encephalitis and has concluded that the experimental evidence is not yet conclusive.

Greenfield[15] has emphasized the marked similarity in the pathologic findings in postvaccinal encephalitis, encephalitis following variola, and encephalomyelitis following measles and considers that the observations support the theory "that both the postvaccinal and other forms of encephalitis which follow eruptive fevers are not directly due to the virus of the preceding fever, but to another virus which is either stimulated to activity, or is directed against the nervous system by the exanthema." Zimmermann and Yannet[16] have described the histopathologic changes of the central nervous system in

two fatal cases of encephalitis occurring during the course of chickenpox and noted that the alterations did not possess any specific or characteristic features which might differentiate them from the pathologic changes noted in variola. After a very careful study, Marsden and Hurst state: "If, therefore, we find the same clinical and pathologic conditions following after a roughly similar 'incubation period' such different predisposing causes as smallpox, measles, antirabic treatment, etc., the conclusion seems inevitable that we are dealing with an independent malady activated by the conditions mentioned and not with one resultant upon some change in virulence or other property of the virus of the primary infection.

Turnbull,[15] on the other hand, has studied the pathology occurring in fatal cases of encephalitis following variola and has concluded that the virus or vaccina is the etiologic factor. After an experimental and pathologic study of the various forms of encephalitis seen following the exanthems, McIntosh[17] decided that the pathology of variola was not only specific in its nature but also in its etiology. He did not concur with those who felt that the various forms of encephalitis developing during the course of the eruptive fevers were due to the activation of a dormant virus. However, from the literature alone one receives the impression that the general opinion is in accord with that as expressed by Greenfield[15] and Marsden and Hurst[11] as quoted above. From the clinical point of view it does appear difficult to understand why the mortality rate and the morbidity rate of the neurologic complications of the exanthems should be so different if the etiology and pathology are the same; for example, the mortality rate in encephalitis complicating measles is 10 per cent, whereas in postvaccinal encephalitis the mortality rate is 58 per cent.

Varicella.—The general tendency is to consider chickenpox as a relatively minor disease and one that more often proves itself to be annoying by virtue of its period of quarantine rather than by the seriousness of its complications. This fact is well illustrated by the short statement of Ker,[18] "There is little to be feared in the way of complications." However, such a statement should not be accepted without consideration, as complications of a most serious nature are not unknown.

The *incidence* with which these complications occur is fortunately low. Bullowa and Wishik[19] reported only five cases of encephalitis and three cases of meningitis occurring among 2,534 patients with chickenpox. Such factors as age, sex, seasonal distribution, and severity of the infection do not appear to exert any direct influence upon the development of disturbances of the central nervous system. As one might expect, secondary infections of the cutaneous manifestations with subsequent development of septicemia may occur, but such complications as these cannot be directly attributed to the etiologic agent causing the primary infection.

Classifications of the various types of neurologic disturbances which may occur during the course of chickenpox have been offered by several observers and have usually been based on either (a) the suggested pathologic lesions or (b) upon the predominant clinical signs and symptoms. As the clinical manifestations of any central nervous system disturbance in which the lesion is of a diffuse nature are seldom comparable to those encountered when there is a well-localized pathologic condition, it becomes apparent that a classification based entirely upon symptomatology cannot be too rigid. The recent classification

offered by Underwood[20] has been based upon both the expected pathologic lesion and the clinical manifestations dividing the cases into "the four main sections of the nervous system—meninges, brain proper, spinal cord, or peripheral nerves—which may be involved." However, in this review no attempt will be made to consider the detailed classifications as the disturbances will only be referred to in general terms.

Encephalitic manifestations occurring in the course of varicella are not uncommon as numerous case reports may be found in the literature.[21-30] The clinical manifestations are usually observed between the fourth and fourteenth day after the appearance of the eruption and are accompanied by muscular weakness and paralysis. The temperature is rarely above 100° F.; stupor and mental dullness are rare, while irritability and delirium are common. During the period of these manifestations definite changes within the cerebrospinal fluid can usually be demonstrated; they consist of an increase in the globulin content and a lymphocytic pleocytosis, the total cell count varying between 5 and 71 cells per cubic centimeter. In the majority of instances the clinical manifestations of the central nervous system disturbance gradually subside, the patient making a complete recovery in the course of eight to ten days.

Complications such as hemiplegia,[31] paraplegia,[32] and ataxia[33-38] developing during the course of the acute phase of the disease or during the period of convalescence have been reported in the literature. Waldman[39] has reported the case of a patient who on the tenth day of her illness developed acute ascending myelitis, and Heller[40] has recorded the occurrence of a fairly typical case of complete transverse myelitis occurring during the course of chickenpox.

Varicella lesions appearing on the cornea have been noted by Terson[41] and Wyler,[42] but impairment of vision or temporary blindness is extremely rare. In 1925 Ratner[43] reported a patient who on the ninth day of illness complained of impairment of vision which became progressively worse and was soon followed by complete loss of sight. Ophthalmoscopic examination revealed marked optic neuritis. Ten days after the onset these changes entirely disappeared, and the patient made a complete recovery.

In the majority of instances the *prognosis* is good although death or a persistence of the clinical manifestations may occur. In a study of 120 cases of which number information concerning 109 patients was available, Underwood found that 12 died, 80 recovered completely, and that 15 recovered with sequelae of some type. As this author points out, some of the sequelae were of little, if any, significance and should these be disregarded, the total number of patients who made a complete recovery would be increased.

Zimmermann and Yannet[16] have described in detail the pathologic changes involving the central nervous system in one of their patients, and as shown by the following quotation, the pathology does not appear to be characteristic: "Widespread, noncharacteristic, degenerative lesions of the ganglion cells with a perivascular destruction of the myelin sheaths in the white matter of the parietal and occipital lobes. There was an extensive phagocytosis of fat in these regions and collections of fat granules in the leptomeninges. Small focal hemorrhages were scattered in the cerebral cortex."

Scarlet Fever.—Complications of the central nervous system are rare in this disease and when they do occur are usually secondary to otitic infections or to infections of the paranasal sinuses. Gordon and Top[44] have recently reported nineteen cases of generalized streptococcus meningitis occurring among 17,311 consecutive hospital patients with scarlet fever. In thirteen of these patients the infection originated from a primary infection of the middle ear, in four from the paranasal sinuses, and in two from suppurative joint infections with subsequent infection of the blood stream. The clinical course of this complication is usually rapid and, except in rare instances, terminates fatally regardless of what form of therapy is employed. In Gordon and Top's group there was only one recovery; Neal and Jones[45] have reported a case with recovery following the intraspinous administration of anti-streptococcus serum. Rosenberg and Nottley[46] collected from the literature forty proved cases of streptococcus meningitis with recovery and to this group added one of their own. Although numerous and sundry types of treatment have been employed, yet no particular one has appeared to be of greater value than another. In brief, one might say that general supportive measures plus removal of the original focus of infection (when possible) and repeated drainage of the cerebrospinal canal have offered as good, if not better, results than the less conservative and more drastic procedures. Specific or would-be specific measures have been rather disappointing.

Encephalitic complications are less frequently encountered than are the purulent infections and, as this statement would infer, are extremely rare. Mornet and Vezin[47] have recently reported a patient who developed the clinical manifestations of encephalitis during the course of scarlet fever as evidenced by stupor, pharyngeal paralysis, coma, and alterations within the chemical and cytologic reactions of the cerebrospinal fluid. This patient made a complete recovery. The patient of Dagnelie and Dubois[48] who developed ataxia, slow speech, and intention tremor, died, and at the postmortem examination diffuse perivascular infiltration, demyelinization, and edema of the brain were noted. Both Rossi[49] and Dubois[50] have cited cases of ataxia following what clinically appeared to be encephalitis, and Van Bogaert[51] has admirably discussed the nervous manifestations that may be observed during the course of scarlet fever. Rolleston[52] had under his care a boy, three and one-half years old, who on the thirty-third day of severe septic scarlet fever developed aphasia and hemiplegia. The boy subsequently made a complete recovery from the aphasia and some improvement from the hemiplegia. The above author has collected from the literature seventy-five cases of hemiplegia following this disease and has suggested four possible etiological factors: (a) cardiac involvement with embolism; (b) uremia; (c) encephalitis; and (d) cerebral atrophy and sclerosis. A case similar to the one reported by Rolleston has recently been noted by Moschini.[53] If we are to accept the statistics which may be obtained from the literature, it would appear that complications involving the central nervous system are rare in scarlet fever and when they do occur usually manifest themselves in the form of purulent infections.

Diphtheria.—The neurologic complications which may appear during the course of, or following, diphtheria[54-58] are so well established and recognized that a discussion of them at this time would simply be repetition. However, a few of the facts relative to these disturbances may be of interest. It has been the general impression, and statistics

would indicate, that a larger number of patients with postdiphtheritic paralysis are encountered today than were encountered before the institution of serum therapy. However, this does not question the efficacy of specific treatment in any way, as the increased incidence can readily be accounted for by: (a) an increase in the total number of cases diagnosed as diphtheria by means of bacteriologic methods (cases which formerly were not recognized); and (b) a larger number of the very severe cases with survival and therefore with a greater opportunity for the development of paralysis.

Although paralysis is more frequently encountered in the severe forms of the disease, its occurrence with mild forms of faucial or even nasal diphtheria is not unknown, and particularly is this true if the disease has been unrecognized and therefore untreated. It would appear that the anatomic location of the infection plays some part in the incidence of the complications as they are more likely to occur in those cases in which there is both faucial and nasal involvement than in those in which the larynx and trachea are involved alone. At this point it might be well to call attention to the fact that paralysis has been observed among those individuals who have had cutaneous infections alone,[59] such as diphtheritic infections of the conjunctiva, umbilicus, penis, or wounds.

The distribution of the paralysis cannot be stated in a categorical manner, for not infrequently the neuritis may entirely subside in one area only to reappear in a remote group of muscles. However, the situation most frequently involved is the soft palate, for here paralysis has been observed as early as the fourth day and often makes its appearance by the end of the second week. Paralysis of the ciliary muscle with resultant loss of the power of accommodation probably occurs second to palatal paralysis as far as frequency is concerned, although there is some question as to whether or not involvement of the lower extremities should not be entitled to this position. Except for cardiac failure, paralysis of the respiratory muscles, either the intercostal muscles or the diaphragm, offers the most serious complication. Fortunately, this type of involvement is not very common as it usually appears only in those instances in which there is generalized paralysis.

Although the diagnosis can usually be established without difficulty, yet not infrequently other diseases may so closely simulate postdiphtheritic paralysis that confusion arises. This is particularly likely to occur when a definite history of infection cannot be obtained or when there has been some vague or indefinite preceding illness. Paralysis occurring as a result of poliomyelitis or in the course of chronic lead poisoning may produce a clinical picture comparable to that of diphtheria. Furthermore, since loss of sphincter control, paresthesia, and analgesia may be present when the lower extremities are involved, one can easily see how tumors of the spinal cord may at times prove confusing. However, the course of the disease with its tendency toward gradual improvement and the inability to demonstrate the presence of other conditions will usually establish the diagnosis.

The prognosis in postdiphtheritic paralysis is usually good, as the general tendency is toward a slow but complete recovery. The respiratory and pharyngeal muscles regain their function at a very early date, although the palatal muscles and the muscles of the extremities may require several weeks or even months before their function returns.

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Round Table Discussion on Allergy in Children

Chairman: Dr. Francis M. Rackemann, Boston, Mass.
Assistants: Dr. Bret Ratner, New York, N. Y.
Dr. Marion B. Sulzberger, New York, N. Y.

CHAIRMAN RACKEMANN.—Allergy may be defined as the abnormal reaction manifested by certain individuals toward certain substances which are harmless for normal persons. This definition includes the two main forms of allergy: the immediate response, urticarial in nature, which determines the pathology and the symptoms of hay fever, asthma and perhaps eczema; and the tuberculin reaction, inflammatory in character, which is closely concerned with the mechanism of many infectious diseases. There is evidence that these two reactions are related and that each one represents a phase in the development of the normal immunity. The immediate urticarial reaction is so characteristic and of such great importance clinically, and the individuals who manifest it comprise such a characteristic group that Coca has conceived of the term "atopy" with "atopic" reactions to "atopens" for the definition of this group.

How do certain people become allergic or atopic? Coca, Cooke, and others believe that heredity plays an important part. This heredity, however, applies more to the capacity for developing a specific reaction than for the particular reaction which occurs. Allergic individuals show as a group, certain general clinical characteristics, as follows:

1. They present the characteristic picture of hay fever, asthma, or eczema, or possibly others.
2. They have some other symptom along with the first.
3. They present positive skin tests.
4. They have a blood eosinophilia.
5. They have a positive family history of allergy.

Allergic or "atopic" individuals have then the capacity to develop sensitiveness. The particular form which this takes depends in most of the cases upon the patient's environment. If there are dogs, cats, feathers, or kapok, he may become sensitive to them. The occupational group in adults is particularly interesting here. In young children, new foods become new points of contact and sensitiveness often appears. Sometimes, however, and particularly in infants, these symptoms arise after the first contact. In these cases Ratner has demonstrated that the specific sensitiveness is transferred through the placenta or the breast milk. It is important, however, to distinguish this maternal transmission of allergens, or of antibodies, as the case may be, from the inheritance of the fundamental allergic state.

Our interest in allergy has so far been concerned chiefly with the diagnosis of the exciting cause. In this, the important method is not the skin test but the history. If true sensitiveness is present, the fact should be demonstrated by showing that symptoms appear on contact and disappear when contact is broken. Histories which include particular dates of changes in environment as well as of new symptoms, and which account for all the time, should contain enough evidence for the diagnosis. Skin tests themselves are interesting, but the discrepancies between skin tests and history are even more interesting. Many patients have skin tests without symptoms while others fail to react even though they are sensitive clinically. In each case, therefore, the skin tests must be correlated with the history so that the picture as a whole will make sense. The technic of skin tests and the choice of method are always important. Sometimes, the scratch test is negative when the intracutaneous test is positive, while in other cases the intracutaneous method shows such irritability of the skin that the results mean nothing. Eye tests, nasal tests, and patch tests are each useful in its special case.

DR. W. C. C. COLE (DETROIT).—Will you say more about the physiologic nature of allergy?

CHAIRMAN RACKEMANN.—How or why sensitiveness develops is unknown. It is a fundamental question. Heredity plays a part. The artificial sensitization which occurs after a dose of horse serum is more easily understood. After ten days or so, antibodies appear in the blood as precipitins and in the skin as a positive skin test. The circulating antibodies are temporary, disappearing within a few days or a week or two, while the skin test persists often for years, and this provides another illustration of positive skin tests which indicate the past history rather than the present illness. However, a latent sensitiveness also exists, and, if another dose of horse serum is given later on, the reaction may be violent. This mechanism is interesting because clinical sensitiveness is often acquired in patients who have the fundamental capacity to develop it. The particular substance to which sensitiveness is directed depends on the patient's contacts. Occupational asthma must arise in this way. One patient has horse asthma; another has orris powder rhinitis according to his or her previous contacts.

DR. RATNER.—Sensitiveness to foods no doubt arises in the same way—by repeated contact.

DR. SULZBERGER.—I agree that the fundamental causes for the varying degrees of susceptibility are still unknown. The quantitative factor in exposure is often evident, but it represents only one factor among the many important and unknown influences which terminate in the production of clinical hypersensitivity. There are many different forms of specific sensitization. Some are acquired regularly and represent a normal response. The regular production of tuberculin sensitivity is an example. In the production of abnormal sensitizations, there are, in addition to hereditary predisposition and the influence of infections, etc., other factors which may reduce resistance or favor sensitization under certain particular conditions, not always understood, but including the time and the duration of exposure of the given individual to the given allergen.

DR. LOUIS DIENES (BOSTON).—No doubt at certain times certain changes occur which predispose to sensitiveness. During infection, such as whooping cough, that part of the system which makes antibodies is much more irritable. Sensitiveness frequently follows infections.

DR. J. C. OVERALL (NASHVILLE, TENN.).—Why is the tuberculin test negative or reduced during and after an illness?

DR. SULZBERGER.—Tuberculin tests apparently become negative under two conditions: (1) during an overwhelming illness, or in a cachectic state—so-called negative anergy; and (2) as a result of progression toward immunity—as seen in the *positive* anergy found in sarcoids, for example. A negative tuberculin reaction is the rule in miliary tuberculosis; but here the skin, as a rule, ceases to exhibit reactions to other substances, such as trichophytin, typhoid vaccine, etc. This is apparently because the macroorganism is overwhelmed by the infection, and the skin has lost all power to react. This is, as I have said, the state of so-called negative or *nonspecific* anergy.

QUESTION.—How long does allergy persist?

DR. RATNER.—As a rule, allergy is not present throughout life. It is evanescent.

CHAIRMAN RACKEMANN.—I do not feel that way. Skin tests persist; symptoms do not. Clinical sensitiveness to particular substances may come and go. Egg sensitiveness is common in infants, rare in adults; and yet skin tests to egg may persist for many years before they fade. The fundamental allergy does, I believe, persist. I think of a boy who had infantile eczema due to eggs; now at the age of eighteen years, he has hay fever. He can eat eggs though his egg test is still there. His capacity to develop sensitiveness to foreign substances in his environment, that is to say, his "allergy," remains with him.

The *diagnosis* of the exciting cause of symptoms is a problem of great practical importance. I would like to emphasize that in my own experience at least the *history* is of far greater value than the skin tests. In taking a history on a patient with hay fever, asthma, or eczema, there are two important points. One is to record dates: the date on which the attack began and the date on which it ended. It is often quite as important to discover the reason for recovery as it is to find the reason for the onset of symptoms. Other events, intercurrent diseases, operations, changes in residence or occupation, must be dovetailed into the history to make a consecutive story, arranged chronologically. The other point is to *account for all the time.* If the patient was free of symptoms for several months or years, the fact is important, and one should ask "why."

DR. SULZBERGER.—It is of the very greatest importance to get time and dates into the history. In the investigation of allergic disease, I should rather dispense with all skin tests than have to work without adequate and careful histories. Only the combination of exact and intelligent history-taking, and exact and intelligent skin testing can produce the optimum results in the search for causal excitants.

DR. OVERALL.—The mother's history may be as important as the child's history—especially the history of her pregnancy.

CHAIRMAN RACKEMANN.—*Skin tests* constitute the drama of allergy. There are several methods and a multitude of materials. The wise selection of each will save time and lead to better results. Skin tests should confirm the history, at least they must be correlated with it, to make sense. In a few instances they will suggest new possibilities and these too must be explained and correlated by the history or by clinical experiments with diet and environment. For routine use, the scratch method is satisfactory in children. Occasionally, the intracutaneous method must be employed, and here the technic is most important. When ragweed will cause a reaction in a dilution of one part in a million, the necessity for chemical as well as bacteriologic cleanliness becomes apparent. Separate syringes, scrupulously clean, each reserved for a particular allergen, must be used.*

*Rackemann and Simon: J. Allergy 6: 184, 1935.

The discrepancy between skin tests and history is often marked. On the one hand, we find positive skin tests which have no clinical significance. As indicated above, they often represent past history. I have patients who formerly had hay fever. Now their symptoms have disappeared, but their skin tests persist. On the other hand, we find negative skin tests in patients who are clinically sensitive. This is harder to prove, but there are clinical examples. If these two extremes are facts, is it not obvious that the interpretation of skin tests is always difficult?

DR. SULZBERGER.—The contamination of syringes is a most important factor of error. This is one reason why the use of scratch tests is preferable for all those who do not specialize in allergy. In quantitative tuberculin testing, in which high dilutions are used, the contamination of syringes becomes apparent at once. No syringe once used for a strong concentration may be employed later for a higher dilution. Traces of tuberculin defy removal, even by washing or sterilization of the glass. Such traces can entirely vitiate the results of tuberculin testing in high dilutions.

DR. W. A. McGEE (RICHMOND, VA.).—Do different extracts, glycerol or saline, for example, require different interpretation?

CHAIRMAN RACKEMANN.—No! Extracts, however made, should show reactions only in sensitive patients, never in normal controls.

DR. McGEE.—Do you test with fungi?

CHAIRMAN RACKEMANN.—In our clinic, tests with extracts of fungi are usually negative. The question reminds me of our recent experiences with kapok, cotton linters, and other vegetable fibers. As first brought out by Milton B. Cohen, we find that extracts of kapok which is new give positive tests in only a few patients. When, however, old used kapok, as found in mattresses and pillows, is employed, extracts give positive tests in a high percentage. This old kapok is moldy; it is the molds which cause the fibers to break down into a fine dust; and our present theory is that symptoms come from the action of the mold (which in itself is harmless) on a substrate of vegetable fiber (which in itself is also harmless). Changes in bedding and sometimes in other furniture or toys result not infrequently in a relief of asthmatic symptoms which is brilliant. Direct inspection by a visit to the house of the patient may lead to dramatic results.

DR. SULZBERGER.—Asthma may be due to many as yet unsuspected causes. As only one example, trichophytin fungi, or monilia fungi, harbored in an insignificant skin or nail focus, may be a cause of perennial attacks. In such (of course, rare) cases, I have seen attacks of typical asthma produced by injections of trichophytin or oidiomycin extracts.

DR. G. F. WEINFELD (RAVINIA, ILL.).—If the skin test for foods is negative, is there any value in elimination diets?

CHAIRMAN RACKEMANN.—Yes, clinical sensitiveness may exist even if the skin test be negative.

DR. J. C. MONTGOMERY (DETROIT).—How often is asthma in children due to food in cases in which eczema does not occur?

CHAIRMAN RACKEMANN.—That is an interesting question because food sensitiveness goes so often with eczema and dusts, with asthma. The opposites, however, do occur, though not commonly.

DR. R. D. HOSTETTER (DAYTON, O.).—Will an intradermal test sensitize the patient and lead to asthma?

CHAIRMAN RACKEMANN.—I know of no specific case, but I can remind you that Hooker found a considerable number of patients tested with toxin-antitoxin mixtures in the Schick test showed later a positive skin reaction to horse serum, and similarly Zinsser, Dienes, Simon, and others, working with both animals and man, have demonstrated the development of a positive skin reaction following repeated intracutaneous injections of the specific proteins, usually foreign serum. Symptoms, however, were not produced.

CHAIRMAN RACKEMANN.—Let us turn to *infection as a cause of asthma.* There are four ways in which infections influence allergy.

1. As Dr. Dienes has said, an acute infection alters that part of the system which makes antibodies. Asthma may often begin after whooping cough, measles, or other acute infection.

2. When allergy is established, the effect of an intercurrent infection will depend upon its severity. As Dr. Sulzberger has already noted in discussing the tuberculin reaction, an acute infection of relative severity may abolish the allergy temporarily. The allergic child who gets measles will lose his eczema or his asthma at the time of the intercurrent disease. This relationship is the principal basis for the use of nonspecific therapy.

3. An acute infection of mild severity like a head cold will aggravate the allergy. There are many children and adults who have positive skin tests to cat hair, kapok, or house dust, for example, and yet no symptoms from them until they "take cold." The new infection brings the sensitiveness to the level of clinical symptoms.

4. "Bacterial allergy" is a common term, which implies that the individual may be sensitive to bacteria directly and have symptoms from the infection itself. Here again, processes both acute and chronic are concerned. There is a small group of individuals who have asthma only twice a year, in spring and fall, in attacks following acute colds. There is no change in their environment or diet; their skin tests show no reaction to common foods or dust. The new cold is the only evident cause of trouble. Another group of patients has mild chronic infections in teeth or sinuses which evidently cause trouble because the asthma is relieved when the focus is removed. A diagnosis of bacterial allergy seems inevitable in both groups.

DR. MONTGOMERY.—Will a tonsillectomy performed in the pollen season set up a sensitiveness?

CHAIRMAN RACKEMANN.—Yes, conceivably. Like new infections, surgical operations or injuries may change the resistance mechanism, so as to modify both the development of sensitiveness and the course of the process after it is established.

Asthma in Children

DR. RATNER.—Time does not permit a lengthy discussion of the problem of asthma in childhood. There are a few points that concern the pediatrician. It is generally conceded that in childhood asthma is, in many instances, preceded by eczema and the general syndrome of exudative diathesis first described by Czerny. As pediatricians, we must be on the alert, for this condition does come on insidiously. It is a mistake, however, to diagnose asthma on the basis of a single isolated attack of dyspnea accompanied by the objective finding of sibilant and sonorous râles. I would never tell a parent that a child has asthma after such an episode. It is only after repeated occurrences of such syndromes that one should begin to think of asthma. Repeated attacks of so-called colds without temperature or repeated attacks of vasomotor rhinitis may be forerunners of asthma. During the period of observation and before any definite diagnosis is made, much valuable data can be obtained by careful questioning with reference to the relation of the illness to particular foods; to environmental factors, such as a particular room, a visit, contacts

with animals, the occupations of parents or other members of the household; and other possibilities that may eventually be pieced together and aid in detecting the actual cause of the allergic attacks if such they are. It might be intimated to the parents that allergy may be the cause of the child's illness.

Much harm is being done to the good name of allergy by a too hasty resort to protein skin tests. Many cases of allergy, diagnosed in an early stage, will give completely negative results at the inception of the disease. It requires repeated attacks to sensitize the skin and make it possible to obtain positive skin reactions. Furthermore, a child should not be subjected to skin tests in the early stages of asthma because in many cases the condition is evanescent and clears up spontaneously. Asthma, therefore, is a condition that should and can be adequately handled and controlled by the pediatrician, and it is only those cases which are complex and intractable that the allergist should be called upon to study thoroughly, which study means hundreds of skin tests, careful analyses of blood and urine, x-ray examinations, and a most careful study of the environment.

I have found that a large percentage of some of the severest cases of asthma give negative reactions to the "ordinary" skin tests but definitely positive reactions to extracts made from substances in the immediate environment of the child. I should like to cite an example: A child had been studied by several allergists and gave negative reactions to all the proteins used; I obtained similar results to the routine proteins, but, when we made extracts, he reacted positively to thirty-odd samples of stuffings from mattresses, pieces of furniture, comforters, and so on. These samples all turned out to be identical in nature, old cotton linters. The incriminating articles were either removed, replaced, or specially treated; the child has been free from asthma for a period of several years, with the exception of two attacks which were directly traced to contact with cotton linters in one instance at school and the second in the family's country home.

DR. A. B. C. HOYER (CINCINNATI).—Dr. Ratner, how did you treat the child?

DR. RATNER.—In these cases the child should sleep on a horsehair pillow, and, if possible, on a horsehair mattress. All the articles containing cotton linters that can be eliminated from the environment are discarded. Those articles of furniture which cannot be discarded should be thoroughly cleansed and reupholstered by covering with an impervious dustproof material, all the joinings being carefully sealed. Egyptian cotton is best, especially if a double thickness is used.

DR. OVERALL.—How about covering with rubber?

DR. RATNER.—Rubber is inadvisable because of the sweating and because it is not durable.

As to the infectious element in asthma, all the work done in the direction of bacterial allergy tends to prove beyond doubt that molds, bacteria, and viruses may very well produce asthma because of the proteins resident in them, for even the virus has recently been shown to be of protein nature. Infectious allergy is brought about by overwhelming contacts with particular parasites which sensitize the individual, and, when these organisms again come in contact with the host, allergic symptoms may result. Repeated invasions of streptococci may produce allergic phenomena in rheumatism or may account for acute upper respiratory infections. Repeated invasions of the tubercle bacillus in time may produce allergic tuberculous phenomena.

DR. S. H. ASHMUN (DAYTON, O.).—Is hypothyroidism common in allergy?

DR. RATNER.—For many years we have studied the ossification centers of all our children and have given thyroid to those children who had delayed ossification. The results in several cases of eczema were truly brilliant. Giving thyroid to certain asthmatic children, I believe, materially helped them in their general resistance.

I do not believe, however, that hypothyroidism per se plays a direct rôle in allergy. Obviously, every allergic patient who shows symptoms of hypothyroidism should receive the benefit of thyroid therapy.

DR. C. D. BUTLER (OAK PARK, ILL.).—Nothing has been said of the *psychic effect*. Does it play a part in the cause of asthma or allergy?

DR. RATNER.—The psyche unquestionably plays a certain rôle in asthma. It is not a primary one, however, and is the result of a "conditioned reflex." I have seen cases in children who have learned to simulate asthmatic attacks. These psychic episodes, which grow out of the desire of the child for attention, must be treated intelligently or else they may become fixed. The same circumstances may cause undue irritation and scratching in cases of eczema and contact dermatitis. Many cases could be cited to show how eczema can be prolonged by such psychic conditioning.

CHAIRMAN RACKEMANN.—No doubt the psyche is important. However, in asthma at least, nervousness and other disturbances of the psyche are more likely effects than causes of the asthma.

DR. SULZBERGER.—All disease processes are, to some degree, dependent upon the general state, and thus to some degree upon the psychoneurogenous mechanisms. There can be no denial that, particularly in reactions due to allergic hypersensitivity, the influence of the psyche may be most evident and, in some instances, may even play a leading part in the production or inhibition of reactions.

DR. F. P. GENGENBACH (DENVER).—How do you explain the fact that some individuals are more sensitive to flea or insect bites than are others?

DR. RATNER.—*The basis for sensitivity to insect bites* is exactly the same as the basis for other sensitivities, namely, that children who have been bitten may under certain circumstances become sensitized, and subsequently insignificant bites may produce marked reactions.

DR. SULZBERGER.—Even the normal skin reactions to insect bites represent an acquired type of skin hypersensitivity; in some instances responsible specific antibodies can be demonstrated by the Prausnitz-Küstner technic. Some of the cases of hypersensitivity to insect bites can be cured by specific hyposensitization measures. I have had several successes with such measures. This entire subject has been carefully studied. Those interested may begin reading up the voluminous literature by first studying the monograph and bibliography of O. Hecht.*

DR. BUTLER.—What part do *physical aspects, such as heat and cold*, play in the cause of asthma?

DR. RATNER.—I believe that heat and cold play a secondary, if any, rôle in the causation of asthma. There are some who assert that heat or cold are primary causes in certain cases. It is my impression that heat and cold may aggravate a particular attack but that the actual cause of the asthma is a sensitivity to some definite allergen.

I have been asked to discuss the question of *food hypersensitivity*. Children become sensitized to foods in one of several ways: The first contact is during fetal life; in the latter months of pregnancy, the mother, eating excessively of certain protein foods, may have enough undigested food proteins in her circulation which readily traverse the highly permeable placenta and thus sensitize the fetus. Second, infants may be sensitized during the newborn period by the passage of undigested protein, particularly milk, through the highly permeable intestinal tract. Third, overfeeding in early childhood may also lead to sensitization. It is, there-

*Hautreaktionen auf die Stiche blutsaugender Insekten und Milben als allergische Erscheinungen, Zentralbl. f. Haut- u. Geschlechtskr. 44: 241, 1933.

fore, of paramount importance to control the diet of the pregnant woman and to introduce foods in early infancy in a partially denatured form and in small amounts, thus establishing a tolerance.

A question has been raised about sensitivity to egg. It is rather interesting that egg white is one of the most common offenders. The reason for this may be due partly to the fact that the molecular weight of egg white is rather small and to a larger extent to the fact that raw egg white escapes digestion and therefore enters the circulation in a native state more frequently than other proteins; this holds true for lactalbumin and possibly other food proteins. Egg white is prevalent in various food preparations so that very often a child may be getting it without the physician's even being aware of it.

DR. OVERALL.—How about using elimination diets as an aid in diagnosis if you do not use them in treatment?

DR. RATNER.—Elimination diets in the diagnosis of food allergy have been popularized by Dr. Rowe and used extensively in the past few years. These diets may be of great value in ferreting out the cause of food allergy in adults, and they are of value occasionally in childhood when the allergy is of a gastrointestinal nature, in which cases negative skin reactions are the rule. Because of the simplicity of the elements that enter into the young child's diet, however, it is of greater value to take very careful histories or to have the mother keep a diary. In the treatment of food allergy, the foods to which the child is sensitive or those that I suspect to be the cause of trouble should be eliminated. In creating a true elimination diet, one must not only remove the particular food itself but be certain to eliminate all forms of food that may contain even traces of the offending protein. At times it is necessary to eliminate such foods as fish, eggs, and nuts from the household because of the possibility of contamination of cooking utensils and other objects used in the preparation of foods.

DR. McGEE.—How about the methods of Dr. B. M. Keston, Dr. Irene Waters, and Dr. J. G. Hopkins, New York, N. Y., for oral desensitization?

DR. RATNER.—These methods have been used by others prior to them. I practically always desensitize my food-sensitive patients orally. Tolerance can be established in about six to nine months; but it is essential for the child to take moderate amounts of the foods to which he has been desensitized quite regularly after the point of tolerance has been reached. In rare instances in which one may not have the cooperation necessary for oral desensitization, I desensitize against foods by injection.

DR. McGEE.—What does one do with a child who is sensitive to rabbit hair?

DR. RATNER.—In such cases eliminate all rabbit hair furs, stuffings of pillows, fuzzy woolens and gloves, felt blankets and hats, and toys covered with rabbit hair. Do not attempt to desensitize toward this fur except in very rare instances in which you may give injections in very minute doses.

CHAIRMAN RACKEMANN.—How young do you start hay fever treatment?

DR. RATNER.—I never start hay fever desensitization in children who are very young. As a rule, pollen tests in children may be done when they give a history of seasonal sneezing, and this can be determined only after the second or third year of life. Such patients may be watched and taken to the seashore for the summer and treated seasonally with tincture of belladonna and an acid-forming diet. If, however, the case becomes pronounced, I then start perennial desensitization. It is necessary to adhere to doses that give practically no reaction. It is important to give very minute injections so as to avoid making the child more sensitive.

DR. McGEE.—Do you use epidermals in desensitizing children?

DR. RATNER.—In the overwhelming majority of cases, I prefer not to, as the wholesale use of injections is not well advised and at times dangerous because severe shock and even death may result. If the epidermal dust in the environment can be reduced or eliminated, it has been my experience that the child gradually becomes tolerant.

The pediatrician has within his power the reduction of the incidence of allergy in childhood by a most careful control of the environment and diet throughout infancy and early childhood. The fundamental principles of infant feeding have been evolved because of allergic reactions that have been variously diagnosed as exudative diathesis, colic, pylorospasm, cyclic vomiting, diarrhea, appendicitis, and anorexia nervosa. The regimens advocated by our most eminent pediatricians with their insistence upon the introduction of new foods in small and gradually increasing amounts, the use of well-cooked foods, boiled milk, evaporated milk, hard boiled eggs, etc., are part and parcel of the armamentarium of the allergist who advocates the use of denatured foods for food-sensitive patients and accomplishes desensitization by the use of gradually increasing amounts. As to the environment, the parent can be taught how to equip the nursery, to avoid such articles as down pillows, stuffed furniture, cotton mattresses, and stuffed toys.

DR. ESTHER B. CLARK (PALO ALTO, CALIF.).—Can any child become asthmatic without a family history of allergy?

DR. RATNER.—Yes, a child can become asthmatic without a family history of allergy. I have just completed a study over a period of many years on the inheritance factor in childhood allergy, and my conclusion is that inheritance per se has not been proved to play a rôle in allergy. We found that approximately 50 per cent of the family histories of allergic children were negative and, conversely, that 50 per cent of the families of *normal* children gave positive allergic histories. I believe that allergy is essentially an acquired condition, the same as infectious diseases. On the one hand, you are dealing with the invasion of an allergen, and on the other hand, with the invasion of a bacterium or virus.

DR. HOSTETTER.—How many tests do you do on household dusts and materials?

DR. RATNER.—The patient is requested to bring samples of all of the pillows and other bedding, stuffed furniture, rugs, clothing, toys, in short, all dust-producing articles in his immediate environment. These samples are placed carefully in separate envelopes and labeled. They are analyzed and an extract of each is made for testing. The number may vary anywhere from a dozen upwards. The most common provocators are old cotton linters, old feathers, rabbit hair, wool, and silk.

DR. L. F. HILL (DES MOINES, IOWA).—Please tell of what value are allergen coverings that are commercially advertised.

DR. RATNER.—Their value depends upon the closeness of the weave of the material. Rubberized materials, as I have already explained, are the least valuable, and Egyptian cotton, the most desirable. Thoroughly cleaned and sterilized horsehair is the most desirable form of stuffing for pillows and mattresses of the allergic patient even if he is sensitive to horse dander.

DR. WEINFELD.—You discussed the inadvisability of feeding raw protein early. Have you seen the development of a sensitivity to egg albumen?

DR. RATNER.—It is inadvisable to give raw proteins in early infancy because there is a tendency for such proteins to escape digestion and enter the circulation through the highly permeable intestinal tract in a native state and thus produce hypersensitiveness. The question of sensitivity to egg albumen has been discussed.

The question of milk sensitivity has also been raised. Experimental studies show that evaporated milk is the best form of denatured milk to use. This conclusion is based on the fact that the whey proteins in evaporated milk are partially de-

natured; practically all milk-sensitive patients are sensitive to the whey proteins, and the casein plays a negligible rôle. Because the whey proteins are not chemically changed but only physically modified, the child in time develops a tolerance to the proteins of raw milk through the use of evaporated milk which, incidentally, can be fed indefinitely.

DR. T. F. REILLY (SPRINGFIELD, MASS.).—How can you say the reaction in cotton linters test is due to old cotton rather than to ordinary house dust?

DR. RATNER.—We are certain that the reaction to old cotton linters is due to age because the patient will give an absolutely negative reaction to fresh cotton linters and a positive reaction to an extract from old cotton linters from a used mattress or other old piece of furniture. Ordinary house dust is of little value for diagnostic purposes; it is important to determine exactly what ingredient in the house dust is responsible for the sensitivity.

DR. F. J. CORPER (CHICAGO).—I have had patients who were sensitive to pollen and who have desensitized themselves only to have the pollen sensitivity returned later. Does not our pollen injection sensitize rather than desensitize the individual?

DR. RATNER.—It is quite true that a pollen case may become desensitized spontaneously, and the condition then recur at a later period. This may result from an exposure to an excessive amount of pollen which induces a new sensitization. I do agree that pollen injections may sensitize rather than desensitize the individual if large enough injections are given to produce a local reaction; it is precisely for this reason that I strongly advocate the use of minimal dosages that are below the threshold of the patient's reactivity. One thus also avoids the danger of systemic reactions.

DR. CORPER.—What are your criteria for determining specific hypersensitivity?

DR. RATNER.—A carefully interpreted positive skin reaction or, in the case which gives negative skin reactions, a carefully interpreted clinical reaction to a specific substance.

DR. G. M. CLINE.—(BLOOMINGTON, ILL.).—Do you not think there is a lot more pollenosis in babies than we think is probable?

DR. RATNER.—I do not believe that there is a great deal of pollenosis in infancy, for as I have already stated it requires several years of contact before pollen allergy is established. For this reason, pollenosis begins to manifest itself as a rule only after the second or third year of life.

DR. H. F. HELMHOLZ (ROCHESTER, MINN.).—What do you think of the value of the eosinophile count as determining the degree of allergy?

DR. RATNER.—In a very careful study on eosinophile counts made on allergic children, we found that there is no direct correlation between the severity of the allergy and the eosinophile count. We found the widest variation in the count, not only in patients having varying degrees of allergy, but we also found a wide variation in the count in the same individual who would have a high count when he was free from allergic symptoms and a low count during the height of an attack. I should say, therefore, that the eosinophile count is of value in the diagnosis of the allergic state but not of its degree.

The Allergic Aspects of Eczema

DR. SULZBERGER.—I shall discuss only infantile eczema, i.e., the common eczematous eruptions in children from one month to two years of age. The first difficulty encountered in all studies and in all discussions of eczema is that this appellation does not constitute a diagnosis, but only a temporary pigeonhole into which, for the present, we place those itching and papulovesicular scaly and erythem-

atous dermatologic odds and ends which await further classification. We shall not progress much further in the study of infantile eczema until investigators realize fully that the conditions thus designated are heterogenous and do not constitute an entity.

It would appear that, even today, the attempt to separate infantile eczemas into at least four categories were fully justified, and, with proper care, should meet with some measure of success. These four categories follow the present four classes of adult eczematous and eczematoid processes:

(1) atopic dermatitis; (2) contact dermatitis (true eczema); (3) seborrheic dermatitis; and (4) unclassifiable forms, which include intertrigos, fungus, and dyshidrotic eczemas, microtic eczemas of other types, etc.

In the first, the atopic group, the site of the hypersensitivity—i.e., what is called the "shock tissue"—is the superficial vascular net of the cutis, just as the vascular reaction is at the basis of urticarial reactions, of vasomotor rhinitis, of hay fever, and in all probability, also of asthma (Matthew Walzer). There is already sufficient clinical, histologic, and immunologic evidence to conclude that in atopic dermatitis the primary phenomenon is subclinical whealing, and that the eczematization and lichenification is usually secondary.

In the second group—the contact type dermatitis, and the one in which allergy has been demonstrated the most clearly—the "shock tissue" is probably predominantly the epidermis itself; and the vascular reactions, although always present to some degree, are considered as accompanying or secondary phenomena (J. Jadassohn, Br. Bloch, Darier, Civatte, W. Jadassohn, and many others).

The third and fourth groups—seborrheic and heterogenous eczematous or eczematized dermatoses—do not merit lengthy discussions here, for in most forms in this group the immunologic alterations are inadequately known, and strict evidence of a leading rôle played by allergy has not yet been submitted.

It must be stated at the outset that, in infantile eczemas, the contact type is surely much less important than in the adult, and I shall not, therefore, discuss this form here. I shall make no further reference to the rare cases of infantile eczema due to true contact allergens, such as simple dyes, chemicals, anesthetics, etc., and shall not discuss the patch test, which is so important in adult eczema; when I refer to "skin tests" in the remainder of this discussion, I mean the scratch or intracutaneous tests, with so-called protein allergens, which produce their wheal and flare reactions.

In addition to the common seborrheic form of dermatitis in infants, the atopic group ranks first in importance. This is the form associated with familial and personal hay fever and asthma, and with atopic dermatitis of childhood and adult life. Dr. Lewis Webb Hill, Dr. A. F. Coca, and I believe and have proposed that this form should be called "atopic dermatitis of infants."

These atopic skin manifestations are based, in all probability, upon true sensitization mechanisms and correspond to hay fever and asthma. Nevertheless, the best management of these cases still consists, first and foremost, in the proper selection of the correct topical remedies and above all, in their correct mode of application and bandaging (just as in all other infantile eczematous, eczematoid eruptions, and in many other dematoses in patients of any age).

From the allergic standpoint, elimination is much more important than skin testing. Skin tests are never capable of bringing absolute proof of the culpability or innocence of a given allergen. Their results simply indicate possibilities which must be verified or excluded by the essential clinical trials of elimination and reexposure. At my hands, the following have eventually proved themselves to be the best and most practical rule-of-thumb elimination measures in infantile atopic dermatitis.

1. Eliminate all important inhalant allergens, and all the important environmental allergens which may in the infant pass through the epidermis and reach the sensitive superficial blood vessels (transepidermal route). This is done by re-

moving all possible dust-formers (pillows, mattresses, overstuffed furniture, carpets, orris-root-containing face powders, and cosmetics, etc.), and all dust-catchers (draperies, hangings, carpets, etc.). Let the child sleep on a cot in a bare room, on sterilized horsehair pillows and mattress (for double safety, these can be covered by so-called "allergen-proof" covers—remember, the baby carriage must here also bear scrutiny).

2. Denature foods as far as possible without harming the state of nutrition. Add vitamins in pure form if necessary. Here the elimination of eggs is important. Milk should be either the dried, evaporated form, or should be boiled for five minutes. Wheat and other cereals must be borne in mind as not infrequent causes. Change the form of source of carbohydrate (for example, from corn sugar to other sugar, or vice versa). See that the child is not overweight. If there is any tendency in this direction, a simple reduction of fat or carbohydrate calories may help the eczema with astonishing rapidity.

Unfortunately, many very important points cannot even be mentioned in this brief discussion. But it must be emphasized once again that correct detective work and elimination are more important than skin tests. The unreliability of skin tests—if one regards them as absolute guides to clinical management—is well illustrated by the fact that from 60 per cent to over 90 per cent of patients with infantile atopic dermatitis give strong wheal and flare reactions to skin tests with egg white and that many of these patients have reagins to egg white. Yet in many of these very same egg skin-test positive infants, the egg white cannot be causing the dermatitis, for in many of them the eczema has appeared without exposure to egg in any form and the dermatitis continues unabated, under protracted elimination of all possible exposure to egg, even under the most absolute and scientific exclusion of egg and egg products, including specially prepared formulas, prepared in special utensils in a laboratory in which no egg has ever been employed.

Furthermore, the administration of egg produces no exacerbation in some of these cases, even though they have the strongest reaction to egg white skin tests and even though they have egg white reagins in high titer. Those interested are referred to the discussion and literature on this subject (Moro, Worringer, and many others), which is mentioned by Hill and me (Arch. Dermat. & Syph. 32: 451, 1935), and in my recent review, "Allergy in Dermatology" (J. Allergy 7: 385, 1936).

It is unfortunate that the differential diagnoses, particularly of the various forms of infantile eczema, are rendered peculiarly difficult, and often frankly impossible, by the following factors which in the adult forms do not usually present the same difficulties:

1. The tendency to weeping and vesiculation on the part of infantile skins, as compared with adults; so that forms which in the adult can be differentiated because they are usually dry and scaly (seborrheic and atopic dermatitis), when they occur in infants often simulate the vesiculating and oozing contact types.

2. Secondary impetiginization and fungus infection, which are so likely to appear in infants; and thus often obscure beyond recognition and underlying original dermatosis.

3. The fact that in older children and adults the external contact with such substances, as feathers (Peck and Salomon), wool, silk, etc., rarely allow penetration to the sensitive cutaneous blood vessels through the adult type of epidermis. (While it is true that cases are seen in which transepidermal penetration takes place even in adults, these are surely the very greatest exceptions.) In infants such transepidermal penetration is the rule; so that external contact even with ordinary protein allergens may often lead to penetration of these allergens through the intact infantile epidermis to the vascular layers beneath and thus produce infantile atopic dermatitis (which, in my opinion, is both in adults and infants usually a form of submanifest whealing, with the shock tissue in the vessels of the upper cutis).

There are, of course, many mysteries still to be solved here, and our immunologic approach is still highly unsatisfactory in the majority of instances. It may be said, today, that the judicious choice and proper application of the correct dermatologic local measures, sometimes combined with a few superficial x-ray treatments, is by far the most successful method in managing most of these cases. It is here to be stressed that superficial x-ray therapy, properly administered and in proper dosage, is absolutely without danger, even in infants as young as six months.

Among the problems in infantile eczema are:

1. The significance of the egg white skin test reaction and of the egg white reagins.

2. Why do many of these cases improve rapidly in the hospital or in some other new environment—even in the house next door—and then rapidly get worse again at home? Is this due to environmental allergens and, if so, to which particular allergens?

3. Why are marked seasonal fluctuations to be observed in some cases? Is it because of pollens, of wool, or of sunlight?

4. What are the rôles of such physical factors as heat, cold, light, etc.?

5. What are the underlying systemic factors, the nervous influences, the hormonal factors, the metabolic, and other factors?

All these and many more questions will perhaps be answered some day. But I cannot emphasize too strongly that for the present, no real progress can be made until we meticulously analyze and subclassify the infantile eczema group, with its various different and essentially nonrelated components, and that we must all, therefore, now strive for greater clarity in differential diagnosis and for ultimate rational subdivision and clear-cut classification and nomenclature.

DR. McGEE.—What is the treatment when patch tests are positive?

DR. SULZBERGER.—In the case of oils, such as plant oils, desensitization is often effective. In the case of other substances, avoidance is, at present, the only practical measure.

DR. McGEE.—If a man is sensitive to wood or the oil out of wood, do you make a patch test with the sawdust?

DR. SULZBERGER.—Yes.

DR. GENGENBACH.—Certain foods like spinach will cause a contact dermatitis around the mouth.

DR. SULZBERGER.—The cases due to foods and with lesions occurring around the mouth are excellent examples of the transepidermal penetration of the allergen which, as I have said, may take place in infants and children. The reaction is often almost immediate, and of wheal and erythema type, followed by secondary eczematization. I also have a healthy respect for egg white skin test reactions, and know that the ingestion of egg may cause severe and even disastrous reactions in some of these cases. These reactions, however, do not occur in all of them. Nevertheless, some infants showing antibodies to egg, and with huge positive skin test to egg, will not improve with removal of egg, and they will not get worse when egg is administered.

DR. RATNER.—I do not agree with this. Children may not get egg in food, but there are other ways of contact, such as contamination of cooking utensils and dishes with egg white used by other members of the family and the ubiquitous presence of egg in many food substances which escape attention.

DR. McGEE.—How about the leucopenic index?

DR. SULZBERGER.—I have no personal experience with this form of test. It is an extremely old method, included among the first known manifestations of allergy (Widal's crisis), but it has heretofore never been considered a useful or reliable means of testing. I should consider it a difficult procedure and, at best,

one which is very hard to evaluate correctly. Under normal conditions, the white count as made by present methods varies widely. It gives no constant and reliable figure.*

DR. McGEE.—Is milk a common cause?

DR. SULZBERGER.—Diet is the answer in only a small number of cases. Only a certain percentage of infantile eczemas is due to sensitization, and again in these only a small percentage is due to foods alone, while a greater proportion can be helped by elimination of both food and environmental allergens. Among the latter, inhalants (silk, wool, feathers, and even pollens, etc.) must be considered.

DR. GENGENBACH.—Where do you place intertrigo?

DR. SULZBERGER.—I place intertrigo among the eczematized and infected processes. It is not a true primary eczema, but here heat, friction, maceration, acid and alkali, fungi, and other microorganisms, plus the proper terrain, are the important factors.

DR. RATNER.—Why do most eczema cases clear up at about one year?

DR. SULZBERGER.—This spontaneous cure between the ages of one and two years is still a mysterious but undoubted fact. Many so-called therapeutic successes are those which have been assayed just at this time of spontaneous remission or during a remission due to change of environment (hospitalization). It is to be remembered, however, that there is often a later recurrence of the dermatitis, as Dr. Lewis Webb Hill and I have pointed out. As a child gets older, it becomes increasingly more difficult to find the cause of his dermatosis. This is due perhaps to the increasing number of factors in the environment and the greater variety of foods. The number of hypersensitivities often increases, and there is usually a shift from foods to inhalant and environmental substances. It is, however, most important in evaluating therapy to realize that in infants and children the majority of clinical food sensitivities disappear spontaneously. There is no real proof of successful active desensitization to foods by any known method in any representative series of cases.

DR. L. M. EARLE (HOLLYWOOD, CALIF.).—I have had good results by giving large doses of raw linseed oil—three tablespoonfuls three times a day. Improvement began in five days. I did observe that most of the babies had loose stools on the treatment.

DR. CORPER.—I used oleic acid on a number of cases but without success.

DR. SULZBERGER.—As I have stated, we cannot evaluate therapy until we take full cognizance of the erratic natural course of infantile eczema, with its inexplicable spontaneous recoveries and its ready favorable response to simple change of environment, hospitalization, and even to changes of season. For all these reasons, we must continually insist upon an adequate control series of cases. Nor can we carry out reliable therapeutic studies until we learn to classify our cases and to separate the various entities now being promiscuously grouped under the head of "infantile eczema." For example, I could well imagine that the unsaturated oil treatment might be useless in contact or atopic dermatitis, and that it might be excellent in seborrheic dermatitis.

CHAIRMAN RACKEMANN.—Gentlemen, I am sure that this discussion could be continued for several hours longer and with profit to everycne here, but I am sorry to say that our time is up. We thank you for your close and critical attention.

DR. BENJAMIN C. HOYER, Cincinnati, Ohio.
DR. NORMAN T. WELFORD, La Grange, Ill.
Recorders

*See editorial in J. A. M. A. 106: 1988, 1936

REPORT OF THE COMMITTEE ON HOSPITALS AND DISPENSARIES

Contagious Disease Hospitals

Part III

Committee on Hospitals and Dispensaries: Dr. Clifford G. Grulee, Chairman; Dr. George F. Munns, Secretary; Dr. Murray H. Bass, Dr. Laurence R. DeBuys, Dr. Henry Dietrich, Dr. Lewis Webb Hill.

REGION I

Forty-four hospitals returned answers to the questionnaire (Table I).

1. *Staff: Professional Staff—Attending Staff.*—Among 41 hospitals which reported as to whether ward patients were under the exclusive care of the attending staff, 23 answered "yes" and 18, "no" (Table II). In reply to whether community doctors may treat patients, 39 reported: 19, an unqualified "yes" and 16 an unqualified "no." In two only may private patients be treated in private rooms. In some cases the private doctor must be approved by the staff of the hospital; in others he need only be a physician in good standing. In one hospital (No. 24) any outside physician may treat a patient.

House Staff.—Forty hospitals replied. Ten have one or more full-time residents or superintendents; 22 have no full-time men; 3 failed to reply. Eighteen of the 40 receive some salary. Of these, several are part-time physicians serving as pathologists or roentgenologists. In some cases only the director is paid. Twenty-four have resident physicians; 1 hospital reports having assistant residents but no resident. Sixteen accept women as resident physicians; 21 pay salaries which vary greatly, the smallest stipend being $30 a month. Some pay $1,200 and $1,500 annually. The highest remuneration is $4,000 for a superintendent.

The qualifications for residents vary greatly. Only three hospitals demand that their residents be graduate physicians; 8 require one year's previous hospital experience; 3 require two years; 2 require three years, 5 require special experience in contagious diseases; 1 is filled by civil service examination. The length of residency varies from four months' service to an indefinite tenure of office. Two hospitals report permanent residents; 1 other reports an incumbent who has been on service thirteen years, another, fifteen years. Ten hospitals offer one year's residency. Only 6 hospitals require the resident to attend dispensary service.

Eight hospitals require the services of assistant residents; 5 accept women; 6 have one each; 2 hospitals require three; and 1 (No. 35) requires twenty-seven assistant residents.

The salary varies from $25 a month to $1,200 a year with maintenance. The qualifications vary from graduation from a grade A school to one year interneship. The length of service in all is one year. In 4 hospitals the assistant resident works in the dispensary. Nineteen hospitals have interneships; 5 hospitals have one interne each; 4 have two; 1 has three; 1 has four; 3 have six; 1 has seven; 1 has eight; 1 has nine; 1 has seventeen; 1 has thirty (No. 35); 15 admit women, and 4 do not.

Seven pay no salaries; the remainder pay from $10 to $50 a month. Most of the hospitals only require graduation from medical school as qualification for interneship though 4 ask for previous hospital service, and 1 requires one year's pediatric service (No. 14).

TABLE I

REGION I—HOSPITALS

KEY NO.	NAME
	Canadian Hospitals
1	Riverdale Isolation Hospital, Toronto, Ont.
2	Hospital Civique of Que.
3	Hospital Pasteur, Montreal, Que.
4	Alexandra Hospital, Montreal, Que.
5	Isolation Hospital, Edmonton, Alta.
6	Strathcona Hospital, Ottawa, Ont.
7	Vancouver General Hospital, Vancouver, B. C.
	Eastern Hospitals, United States
8	Garfield Memorial Hospital, Washington, D. C.
9	Doris Memorial Hospital (Wilmington General), Wilmington, Del.
10	Scranton Municipal Hospital, Scranton, Pa.
11	Hartford Municipal Hospital, Department of Communicable Diseases, Hartford, Conn.
12	Portland Isolation Cottage, Portland, Me.
13	Sydenham Hospital, Baltimore, Md.
14	Children's Hospital, Boston, Mass.
15	The Board of Health Hospital, Brookline, Mass.
16	Haverhill Contagious Hospital, Haverhill, Mass.
17	New Bedford Isolation Hospital, New Bedford, Mass.
18	Health Department Hospital, Springfield, Mass.
19	Belmont Hospital, Worcester, Mass.
20	Manchester Isolation Hospital, Manchester, N. H.
21	Essex County Hospital for Contagious Diseases, Belleville, N. J.
22	Hudson County Contagious Diseases Hospital, Laurel Hill, Secaucus, N. J.
23	Trenton Municipal Hospital, Trenton, N. J.
24	Municipal Hospital for Contagious Diseases, Camden, N. J.
25	Passaic Municipal Hospital, Passaic, N. J.
26	Municipal Hospital, Atlantic City, N. J.
27	Bide-A-Wee Hospital, Watertown, N. Y.
28	Jefferson County Hospital for Contagious Diseases, Calcium, N. Y.
29	Schenectady Isolation Hospital, Schenectady, N. Y.
30	Richmond Borough Hospital for Contagious Diseases, Staten Island, N. Y.
31	Queensboro Hospital, Jamaica, N. Y.
32	Syracuse City Hospital, Syracuse, N. Y.
33	Niagara Falls Municipal Hospital, Niagara Falls, N. Y.
34	Willard Parker Hospital, Manhattan, N. Y.
35	Strong Memorial Hospital and Rochester Municipal Hospital, Rochester, N. Y.
36	Binghamton City Hospital, Binghamton, N. Y. (general hospital)
37	Albany Hospital, Albany, N. Y.
38	Lakeview Hospital, Erie, Pa.
39	Municipal Hospital for Contagious Diseases, Pittsburgh, Pa.
40	Philadelphia Hospital for Contagious Diseases, Philadelphia, Pa.
41	Contagious Disease Hospital, Wilkes-Barre, Pa.
42	Municipal Hospital, Johnstown, Pa.
43	Charles V. Chapin Hospital, Providence, R. I.
44	Carpenter Isolation Hospital, Holyoke, Mass. (hospital closed)

The length of interneship is under six months in 6 hospitals, six months in 4 hospitals, one year in 6 hospitals, and indefinite in 2.

Educational Activities.—Thirty-seven hospitals replied to this section of the questionnaire (Table III). Only 9 are connected with medical schools: University of Toronto, McGill University, Johns Hopkins University, and the University of Maryland, Harvard University, Syracuse University Medical School, University of Rochester, Albany Medical College, University of Pittsburgh, Tufts Medical School. Clinics are given for medical students in 15 hospitals and students are on ward rounds in 13.

TABLE II

STAFF

HOSPITAL	WARD PTS. EXCLUSIVE CARE DRS. ON ATTENDING STAFF	COMMUNITY DRS. ALLOWED SEND PTS. AND TREAT THEM	QUALIFIED DRS. ALLOWED TO SEND PTS. AND TREAT THEM	PEDIATRICS				OTOLARYNGOLOGY				ORTHOPEDICS			
				NO. SPECIALISTS ATTENDING STAFF	MONTHS ON DUTY EACH YEAR	VISITS AT REGULAR INTERVALS	ON CALL ONLY	NO. SPECIALISTS ATTENDING STAFF	MONTHS ON DUTY EACH YEAR	VISITS AT REGULAR INTERVALS	ON CALL ONLY	NO. SPECIALISTS ATTENDING STAFF	MONTHS ON DUTY EACH YEAR	VISITS AT REGULAR INTERVALS	ON CALL ONLY
1	Yes	No treatment	Licensed and good standing	Yes			Yes	Yes	12	Yes					
2	Questionnaire not returned														
3	Yes	No		2	Full			2	Full						1
4	Yes	No treatment	Private cases	Yes	Yes	Yes		Yes	Yes	Yes		Yes	Yes	Yes	
5	No	No	Recommendation and appointment	4	3		Yes	6	2		Yes				
6	No	Yes		2	12		1	2	12		Yes				
7	No	Yes		Yes			Yes	Yes			Yes	Yes			Yes
8	Yes and consulting staff	Acceptable to staff	Class A hosps.	2	12	Yes	Yes	8	3	Yes	Yes	4	6	Yes	Yes
9	No	Yes		3	4	Yes		3	4	Yes		1	12		Yes
10	Yes	Yes		Yes			Yes	Yes			Yes	Yes			Yes
11	No	Yes													
12	No	Yes		No				No				No			
13	Yes	(Private rooms)	Good standing	Yes			Yes					1			Yes
14	No	No treatment	Approved by committee	Yes		Yes		Yes		Yes		Yes		Yes	
15	Yes	No treatment						Yes			Yes				
16	Yes	Yes		Yes			Yes	Yes			Yes				
17	No staff	Yes	No qualification												

TABLE II—CONT'D

18	No	Yes		Yes	Rotating	Yes		Yes			Yes	Yes			Yes
19	No	Yes	Good standing	Yes			Yes	Yes			Yes	Yes			Yes
20	No staff														
21	Yes	No		8	3	Yes		14	2	Yes		5	3		Yes
22	Yes	No treatment		Yes			Yes					Yes			Yes
23	Yes	(Private rooms)						Yes	Full		Yes	Yes	Full		Yes
24	Yes	No treatment	All Drs.	Yes 1	Full		Yes	Yes 1	Full		Yes	1	Full		Yes
25	Yes	No		1		Yes		3			Yes	1			Yes
26	Yes	No	None	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes
27	No staff	Yes													
28	No	Yes													
29	Yes	Yes		1	12	Yes	Yes	1	12	Yes	Yes	1	12	Yes	Yes
30	Yes	No		No				2	Full	Yes	Yes	1	Full	Yes	Yes
31	Yes	No						2			Yes	2			Yes
32	No	Yes		1 to 6	Full 2 mo. each	Yes		4	3 mo. each			3	4 mo. each	Yes	
33	No	Yes					Yes				Yes				
34	Yes	No		34	3	Yes		8	5	Yes		2	12		Yes
35	Yes	No													
36	Yes		Approval of attending staff	Yes	Full	Yes		Yes	Full	Yes		Yes	Full	Yes	
37	No staff	Yes													
38	Yes	Yes, restriction on treatment						1	Full		Yes				
39	Yes	Yes, no treatment		4	Full		Yes	7	4		Yes	3	Full		Yes
40	No	Yes	Good standing	Yes			Yes	Yes			Yes	Yes			Yes
41	No	Yes	None	2	6	Yes		2	6		Yes	1	12		Yes
42	Yes	No		16	3	Yes		7	3	Yes		3	4		Yes

TABLE II—CONT'D

	OPHTHALMOLOGY				SURGERY				NEUROLOGY				PSYCHIATRY			
HOSPITAL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL
1					Yes			Yes					Yes			Yes
2		No reply														
3	2	Full			1	Full						Yes				Yes
4	Yes	Yes	Yes	Yes	Yes	Yes	Yes		Yes	Yes	Yes		No			
5	6	2		Yes	4	3		Yes	2	6		Yes	1	12		Yes
6	1	12		Yes	2	12		Yes								
7	Yes			Yes	Yes			Yes	Yes			Yes	Yes			Yes
8	3	12	Yes	Yes	14	3	Yes	Yes	3	12	Yes	Yes	No			
9	1	12		Yes	3	4		Yes	1	12		Yes	1	12		Yes
10	Yes				Yes			Yes	Yes			Yes	Yes			Yes
11																
12	No				No				No				No			
13					Yes 2			Yes	2			Yes				
14	Yes			Yes	Yes		Yes		Yes		Yes		Yes		Yes	
15					Yes			Yes								
16	Yes			Yes	Yes			Yes	Yes			Yes	Yes			Yes
17																
18	Yes			Yes	Yes			Yes	Yes			Yes	Yes			Yes
19	Yes			Yes	Yes			Yes	Yes			Yes	Yes			Yes

TABLE II—CONT'D

20									Neurol. and				Psychiat. and			
21	6	4		Yes	15	3	Yes		psychiat. 3			Yes	neurol. 3			Yes
22	Yes			Yes	Yes			Yes								
23	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes
24	1	Full		Yes	1	Full		Yes								
25	1			Yes	4			Yes	1				No			
26	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes
27																
28																
29	1	12	Yes	Yes	1	12	Yes	Yes	1	12	Yes	Yes	1	12	Yes	Yes
30	By otolaryngologist				1	Full	Yes	Yes								
31	1			Yes	4			Yes	1			Yes				
32	1			Yes	4	6 mo. each	Yes		2	6 mo. each		Yes				Yes
33				Yes				Yes								
34	2	12		Yes	5	8		Yes	3	12		Yes				
35																
36																
37																
38	Yes	Full	Yes		Yes	Full	Yes		Yes	Full	Yes		Yes	Full	Yes	
39																
40																
41	2	Full		Yes	2	Full		Yes	2	Full		Yes				
42	Yes			Yes	Yes			Yes								
43	1	12		Yes	2	6										
44	4	6	Yes		2	12		Yes	10 and 3 psych.	6	Yes		See neurology			

TABLE II—CONT'D

HOSPITAL	PSYCHOLOGY				DERMATOLOGY				DENTISTRY				ORAL SURGERY			
	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL
1	Yes			Yes					Yes			Yes				
2	No reply															
3				Yes								Yes				Yes
4	No				Yes	Yes	Yes		No				Yes	Yes	Yes	
5					1	12		Yes	12	1		Yes				
6					1	12		Yes								
7	No				Yes			Yes	Yes			Yes	Yes			Yes
8	No				3	12	Yes	Yes	3	12	Yes	Yes	1	12	Yes	Yes
9	1	12		Yes	1	12	Yes		2	6		Yes	2	6		Yes
10	Yes			Yes	Yes			Yes	Yes			Yes	Yes			Yes
11																
12	No				No				No				No			
13					1			Yes	1			Yes				
14	Yes		Yes		Yes		Yes		Yes		Yes		Yes		Yes	
15									Yes			Yes	Yes			Yes
16									Yes			Yes	Yes			Yes
17																
18	Yes			Yes	Yes			Yes	Yes			Yes	Yes			Yes
19					Yes			Yes								

TABLE II—CONT'D

20																
21	No				7	3	Yes		O.S. & O.H. 10			Yes	Dent. & O. H. 10			Yes
22					Yes			Yes	Yes			Yes				
23	No				Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes
24									1	Full		Yes				
25	No				1				1				1			
26	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes
27																
28																
29					1	12	Yes	Yes	1	12	Yes	Yes	1	12	Yes	Yes
30																
31					1			Yes	1			Yes				
32					2	6 mo. each		Yes	2			Yes				Yes
33																
34					3	12		Yes	1	11	Yes					
35																
36																
37																
38	Yes	Full	Yes		Yes	Full	Yes		Yes	Full	Yes		Yes	Full	Yes	
39																
40																
41					1	Full		Yes	1	Full		Yes				
42									Yes			Yes				
43					1	12		Yes								
44					4	3	Yes		1 & O.S.	12	Yes		See dentistry			

TABLE II—CONT'D

	ORAL HYGIENE				REONTGENOLOGY				PATHOLOGY				ALLERGY			
HOSPITAL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL	NO. SPECIALISTS	ON DUTY	VISITS REGULAR	ON CALL
1																
2																
3					1	Full			1							Yes
4	No				Yes	Yes	Yes		Yes	Yes	Yes		No			
5					2	12		Yes	2	12		Yes	2	6		Yes
6	1	12		Yes					1	12		Yes				
7	Yes			Yes	Yes			Yes	Yes			Yes	No			
8	1	12	Yes	Yes	3	Full	Yes	Yes	3	Full	Yes	Yes	No			
9	2	6		Yes	1	12		Yes	1	12	Yes		4	3	Yes	
10	Yes			Yes	Yes			Yes	Yes			Yes	No			
11																
12	No				No				No				No			
13					1			Yes	2			Yes				
14	Yes			Yes	Yes		Yes		Yes		Yes		Yes			Yes
15					Yes			Yes	Yes			Yes				
16					Yes			Yes	Yes			Yes				
17																
18					Yes		Yes		Yes		Yes		Yes			Yes
19					Yes			Yes								
20																
21	Den. & O. S. 10			Yes	1	Part-time salary			Path. & Allergy 1	Part-time salary			Allergy and Path. 1	Part-time salary		

TABLE II—CONT'D

22					Yes			Yes							
23	No				Yes	Full		Yes	Yes	Full		Yes	No			
24									1	Full		Yes				
25	No				No				No				No			
26	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes	Yes	Full		Yes
27																
28																
29									1	12	Yes	Yes				
30					1	Full	Yes	Yes								
31					2			Yes	2			Yes				
32					1	Full	Yes	Yes				Yes				Yes
33								Yes								
34					2	12	Yes									
35																
36																
37																
38	Yes	Full	Yes		Yes	Full	Yes		Yes	Full	Yes					
39																
40																
41					1			Yes								
42					Yes			Yes	Yes			Yes				
43																
44					1	12	Yes		1	12	Yes					

TABLE III

STAFF—EDUCATIONAL ACTIVITY

HOSPITAL	CONNECTED WITH MEDICAL SCHOOL	NAME	MEDICAL STUDENTS		PHYSICIANS				
			CLINICS GIVEN	WARD ROUNDS OR WALKS	CLINICS OPEN	NUMBER AND CHARACTER	WARD ROUNDS OR WALKS OPEN	NUMBER AND CHARACTER	DEFINITE COURSES IN POST-GRADUATE
1	Yes	U. of Toronto	Yes	Yes	Yes		Yes		Yes
2	Not returned								
3	No		No		On demand				No
4	Yes	McGill Medical School	Yes	Ward rounds	Yes	1 a week	Yes	1 a week	No
5	Yes	Alberta Univ.	Yes	Yes			Yes	Weekly rounds	No
6	No		No	No	May attend	10-15			No
7	No		Yes		Yes				No
8	No		Yes	Yes	Yes	Every branch also conference and presenting papers	Yes	Staff	No
9	No								No
10	No		No		Yes	Occasional	Yes	Daily	No
11	No		No		No		No		No
12	No		No	No	None		None		No
13	Yes	By agreement Johns Hopkins and U. of Md.	Yes	No			Yes		No
14	Yes	Harvard Medical School	Yes	Yes	Yes	Weekly	Yes	Weekly	No
15	No		No	No					No
16	No				No		Yes		No
17									
18	No		No	No	Yes	T. B. for exam. and diagnostic	Yes	Men on service only. Others are courtesy	No

TABLE III—CONT'D

19	No		Yes	Yes	Yes		Yes		
20									
21	No				No				No
22					No				No
23	No		No	No	No		No		No
24	No		No		No		Yes		No
25	No				No				No
26	No		No	No	No		Yes	Not regular	No
27	No		No						No
28									
29	No						Yes		
30	No		No	No	No		No		No
31	No				Yes	1 a yr. Auspices County Med. Soc.	No		
32	Yes	Syracuse U. Med. School	Yes	Yes	No		Yes		No
33	No		No	No	No		No		No
34	No		Yes	Yes	No		No		No
35	Yes	U. of Rochester Medical School	Yes	Yes	Yes	Practitioner 1 a month	Yes	Not regular	Yes
36									
37									
38	Yes	Albany Medical College	Yes	Yes	Yes	Daily	Yes	Daily	No
39	No				No		No		No
40	Yes	U. of Pittsburgh, Pa.	Yes	Yes				As desired by individual practicing Dr.	No
41	No		Yes	Yes	No		No		No
42	No		No	No	No		Yes	Anyone interested welcome	No
43	No				No		Yes	Daily	No
44	Yes	Tufts Medical School	Yes	Yes	Not regularly	Allowed to visit clinics	Not regularly	Allowed to visit	No

TABLE III—CONT'D

HOSPITAL	LIMITED TO	P. G. ENROLLMENT LAST YEAR	P. G. ENROLLMENT THIS YEAR	LENGTH OF COURSE	HOURS	INTERNES			
						CLINICAL CONFERENCES HELD	HOW OFTEN	PATHOLOGICAL COFERENCES HELD	HOW OFTEN
1								No	
2									
3						Yes	Monthly	Yes	Monthly
4						Yes	Weekly	Yes	Occasionally
5						Yes	Monthly	Yes	Monthly
6						Yes	Weekly	No	
7									
8						Yes	Monthly	Yes	Weekly
9									
10									
11						No		No	
12						No		No	
13						Yes	On interesting cases	Yes	On interesting cases
14						Yes	Daily	Yes	Weekly
15									
16									
17									
18									
19									
20									

TABLE III—CONT'D

21						Yes	3 times a week	Yes	Occasionally
22						None			
23						Yes	Monthly	No	
24						No			
25						No			
26						At A. C. Hosp. only		At A. C. Hosp. only	
27									
28									
29									
30						No		No	
31						Yes	Monthly	No	
32						No			
33									
34						Yes	Monthly	Yes	Monthly
35	50	50		1 wk.	9-5	Yes	Weekly	Yes	Weekly
36									
37									
38						Yes	Weekly	Yes	Weekly
39						No		No	
40						Yes	Daily	No	
41						No		No	
42						No		No	
43									
44						Yes	Monthly	Yes	Monthly

TABLE IV

STAFF (SPECIAL INFORMATION)

HOSPITAL	MEDICAL LIBRARY	NUMBER OF VOLUMES	FACILITIES AVAILABLE TO STAFF	DESCRIBE	RESEARCH IN COM. DISEASES ENCOURAGED	HOW LONG ALL CASES KEPT IN OBSERVATION	HOW LONG MAY THEY BE KEPT	PROCEDURES BEFORE ADMISSION TO WARDS
1	No				Yes	Diagnosed	Indefinite	
2								
3	At St. Luke Hosp.		Yes		Yes	15-20 days	Full time	Thorough exam. by resident
4	Yes	350	Yes	At McGill Med. Library	Yes	3 wk.	3 wk.	Examination
5	Yes		No		No	Varies	Definite diagnosis is made	
6	Yes	50	No		Yes	2 wk.		Throat, vaginal, careful physical
7	Yes		Yes		Yes	3 wk.	As long as desired	Examination by interne
8	Yes		Yes		Yes		As required	Isolation, etc.
9	Yes	125			Yes	24 hr.	21 days	
10	No		No		No	2 days	Indefinite	Consultation and clinical lab.
11	Yes	75	Yes	Experimental work for treatment comm. dis.	Yes	No observation ward		
12	No		No			None		None
13	Yes	200	No		No	None (plan for near future)	In isolation full time	Examination
14	Yes		Yes		Yes	None		
15	No		No		Yes	None		
16	No		Yes	Municipal Hosp. Lab., X-ray, etc.	No	Until positive diagnosis	As necessary	Positive diagnosis

TABLE IV—CONT'D

HOSPITAL	MEDICAL LIBRARY	NUMBER OF VOLUMES	FACILITIES AVAILABLE TO STAFF	DESCRIBE	RESEARCH IN COM. DISEASES ENCOURAGED	HOW LONG ALL CASES KEPT IN OBSERVATION	HOW LONG MAY THEY BE KEPT	PROCEDURES BEFORE ADMISSION TO WARDS
17								
18	No				No	1 wk.	As necessary	As follows
19	Yes	100	Yes	More when new bldg. is completed	No	None		
20								
21	Yes	150	Yes		Yes	None		
22	No		Yes		Yes	24-48 hr.	48 hr.	As follows
23	Yes	30	Yes	Clin. lab.	Yes	As needed	As needed	
24	Yes	40	No		Yes			In rooms after history and examination
25	No							
26	In A. C. Hosp.		No		No	None		
27	No					None		Exam. by attending physician
28								
29	Available nearby				Yes			
30	No		No		No	None		Diagnosis confirmed by board of health
31	No		No		Yes	Until diagnosed	Termination of case	Seen by health office
32	No		Clinical and statistical		Yes			Not positively diagnosed go in individual rooms

TABLE IV—CONT'D

HOSPITAL	MEDICAL LIBRARY	NUMBER OF VOLUMES	FACILITIES AVAILABLE TO STAFF	DESCRIBE	RESEARCH IN COM. DISEASES ENCOURAGED	HOW LONG ALL CASES KEPT IN OBSERVATION	HOW LONG MAY THEY BE KEPT	PROCEDURES BEFORE ADMISSION TO WARDS
33	No		No staff		No	24 hr. to 7-10 days	Indefinitely	
34	Yes	187	Yes	If not large expenditures collaborate with Health Dept.	Yes	At least 3 wk.	As necessary	Examination
35	Yes	35,000	Yes		Yes	No observation wards	Individual precaution at least 3 days	Exam. history
36								
37								
38	Yes		Yes	In all depts. of Med. School	Yes	Until diagnosed	Diagnosed or duration of disease	
39	No					24 hr.		
40	No		Yes	Limited lab. services	No	No observation		
41	Yes	85	Yes		Yes	Indefinitely	Indefinitely	Report from Dept. of Public Health
42	No		No		No	All private rooms		
43	No		No		No	Not at all		Admitted directly to wards
44	Yes	1,000	Yes	Clinical cases and records of 25 yr.	Yes	Indefinitely	Indefinitely	Physical exam. culture

(Table IV continued on p. 836.)

Special Information.—Thirty-three hospitals answered this section of the questionnaire (Table IV). In regard to the admission of patients, 13 answered that they have observation wards. Of the remainder several answered that they use the observation ward merely until the case is diagnosed; 5 keep the patient under observation for three weeks; 1, one week; 1, two weeks. Several merely answered "until diagnosed"; 5 kept patients under observation three weeks; 1, one week; 1, two weeks; 5, only twenty-four to forty-eight hours. Most hospitals keep the patient under observation until the diagnosis is positive.

Procedure before admission to wards varied greatly. Eighteen answered this question. In 4 cases the resident admits the patient; in 4 the diagnosis must be confirmed by the board of health; 31 require a throat culture for diphtheria on admission; 3 take this only if there is a suspicion of the disease; 19 take routine throat cultures for hemolytic streptococcus. Four replied that cultures are taken "if indicated." As to vaginal smears for gonorrhea, in 2 hospitals they are made routinely; in 1, only in girls under fifteen years; in 1, in girls under twelve years. Five reply "no," and the remainder, "culture only if indicated" or "on request."

Routine tuberculin tests are done in 9 hospitals; Wassermann tests, in 12; Schick tests, in 16; Dick tests, in 12. Routine blood counts are done in 18 institutions. History is taken in all and complete physical examination in all but 1.

Superintendent.—Twenty-eight hospitals are situated in better class districts; only 1 is in a tenement section. Five are in business districts; 7 are in noisy areas, near elevated railroads or noisy factories. Thirty hospitals are located on smooth paved streets; 1 is on a gravel road. The variation in the importance and size of these institutions is shown by the number of their buildings. Thirty-seven answered this section of the questionnaire; of these, 14 reported one building each; 13 reported two to five buildings; 8 reported five to fifteen; 1 reported nineteen, and 1 twenty-seven buildings (Table V). Some of the structures are very old, one dating back to 1876, but the great majority were constructed during the last twenty years. Fourteen have only one or two floors, but many are large with pavillions of six stories. Twenty-five hospitals report that their buildings are fireproof; 8 that they are not; 25 replied as to firestairs in the walls; of these 11 have them, and 14 have not. Twenty have outside fire escapes.

The cost of the buildings varies with the size of the institution, running from $3,250 to $5,000,000.

As to total bed capacity, 37 replied. The smallest institution has 12 beds, the largest, 907; 8 hospitals have over 250 beds; 14 have 50 beds or less. Thirty have cribs for children under two years, varying in number from 3 to 600.

Thirty-three hospitals answered that they have wards varying in number from 1 to 59. Of those replying, 25 had cubicles, and 8 did not; 5 hospitals report all their cases in cubicles, 1 having 310 bed capacity and another 155.

Thirty-five hospitals replied to the question of exclusion of certain contagious diseases; 20 accept any contagious disease; 5 exclude smallpox; 2 exclude erysipelas; 5 exclude gonorrhea and syphilis; 1 excludes pertussis; 2 exclude typhoid; 2 exclude diphtheria. Some exclude measles and chickenpox, unless complicated.

Contagious Disease Wards.—Thirty-two replied, all of which hospitals have wards but 1 (Table VI). The total number of beds is 2,511. The number of beds per ward varies from 1 to 50. Six have cubicles, 1 of which has as many as 297; 27 have separate rooms, about 225 beds in all. Thirty wards have sunlight; 22 have outdoor porches. In 21 hospitals adults and children are put in the same ward.

Admissions.—The number of cases of children's diseases handled by these hospitals varies, of course, according to size (Table VII). One hospital admitted 66, 60, and 69 cases in 1932, 1933, and 1934, respectively. The largest institution

TABLE IV—CONT'D

HOSPITAL	THROAT CULTURE		VAGINAL SMEARS FOR GONOCOCCI	VON PIRQUET OR MANTOUX	WASSERMANN OR KAHN	SCHICK TEST	DICK TEST	ROUTINE BLOOD COUNTS	URINALYSIS	HISTORY TAKEN	COMPLETE PHYSICAL EXAMINATION MADE
	B. DIPH.	HEMOLYTIC STREPTOCOCCUS									
1	Yes	Yes	Yes	Not always	When indicated	Yes	Yes	Yes	Yes	Yes	Yes
2											
3	If judged useful	If useful	If useful	If useful	If useful	If useful	If useful	Mostly	Admission and 1 or 2 a week	Yes	Yes
4	2 on admission	No	Occasionally	Occasionally	No	Yes	No	Yes	Every other day	Yes	Yes
5	Yes	Yes	Yes	Mantoux in T. B. suspect cases	Yes	Yes	No	Yes	Yes	Yes	Yes
6	Yes	No	Yes	When indicated	Not routine	If doubtful	If doubtful	If indicated	Biweekly	Yes	Yes
7	Yes		Yes				Yes	Yes	Yes	Yes	Yes
8	Yes	Yes	Yes		Yes	Routine	Routine	Routine	Routine	Routine	Routine
9	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
10	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes
11	Yes	Yes	Yes	When indicated	When indicated	No	No	Yes	Routine	Yes	Yes
12	Yes	Not routine	No	No	No	No	No	No	No	Yes	No
13	Yes	Not routine	On girls under 12 yr.	Not routine	Not routine	Not routine	Not routine	Yes	Yes	Yes	Yes
14	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
15											
16	Yes		Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

TABLE IV—CONT'D

17											
18	Yes	Yes	If suspicious		Yes			Yes	Yes	Yes	Yes
19											
20											
21	Yes	No	Yes	Not routine	Not routine	Not routine	Not routine	Yes	Yes	Yes	Yes
22	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
23	Yes	Yes	If requested	No	No	No	No	If advisable	Yes	Yes	Yes
24	Yes	Occasionally	If indicated	No	No	No	No	No	Yes	Yes	Yes
25	Yes	Yes	Yes	Yes		No	No	No			
26	If indicated	No	Yes	No	No	No	No	No	If necessary	Yes	Yes
27	No	If requested	No	No	No	No	No	No	No	Yes	Yes
28	Only on request of attending physician. Then at lab. of General Hosp.										
29	Yes	Yes	Yes		When suspicious	Yes	If in diagnosis		Yes	Yes	Yes
30	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Not always	Yes	Yes	Yes
31			Yes	When necessary	When necessary	Yes	Yes	No	Yes	Yes	Yes
32	Yes	Yes	Yes	Not regularly	Not regularly	Yes	Yes	Yes	Yes	Yes	Yes
33	Yes	Yes							Yes	Yes	Yes
34	Yes	On order	Yes	On order	On order	Yes	Yes	Yes	Yes	Yes	Yes
35	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
36											
37											
38	Yes		When indicated	Yes	Yes	Yes		Yes	Yes	Yes	Yes
39	Yes	No	No	No	No	No	No	No	Yes	Yes	Yes
40											
41	Yes	Yes	Yes			Yes		Yes	Yes	Yes	Yes
42	Yes	Yes	Yes	When indicated	Yes	When indicated	When indicated	Yes	Yes	Yes	Yes
43	Yes	Yes	No	No	If indicated	No	No	No	Yes	Yes	Yes
44	Yes	Yes	Females under 15 yr.	When indicated	When indicated	Whea indicated	When indicated	When indicated	Yes	Yes	Yes

TABLE V

SUPERINTENDENT (GENERAL)

HOSPITAL	NUMBER BUILDINGS IN PLANT	DATE OF CONSTRUCTION	DATE OF ADDITIONS	NUMBER OF FLOORS	FIREPROOF	FIRE STAIRS IN WALLS	OUTSIDE FIRE ESCAPE	COST OF BUILDINGS (NOT LAND)
1	6	1891-3-2, 1910, '22, '27		3	1 yes 4 no	Yes	Yes	
2								
3	2	1933		Main—6, Nrs. hm.—4	Yes		Yes	$1,500,000
4	3	1906, '19, '14		3	Yes	No	Yes	$460,000
5	1	1923		3	Yes		Yes	
6	2	1903, '21	To hosp. 1910, '20	4, 3	No	Yes	Yes	$250,000
7	1	1925, '26		4	Yes	Yes		$250,000
8	14	1884, '87, '92, '93, '98 (3), 1902, '09, '12, '15, '25, '33		5—2 fl., 2—4 fl, 6—3 fl., 1—5 fl.	Built since 1909	Recent	Yes	
9	1	1931		3	Yes		2	$180,874.20
10	6	1915		1	No			?
11	5	4 bldg. 1914	1 fl. 1920	3—1 fl., 2—2 fl.	No	No	No	$99,906.83 $44,906.13 (addit.)
12	1	1919		2	No	No		
13	1	1923		3	Yes	Yes	No	
14	1	1931		4	Yes	Yes		$340,000
15	5			1—3 fl., 4—1 fl.	Yes			?
16	1	1914		3	Semi	No	Yes	$41,210
17								
18	4 2 in use	Main bldg. 1929		2	Yes	Yes		$100,000
19	12 1 T.B.	4—1896, 2—1907, 2—1913, 1—1921, 2—1925	1907		Yes			Approximately $1,500,000

TABLE V—CONT'D

20	4			2	Yes			
21	13	7—1908, 2—1922, 1—1929, 3—1932	Power plant and laundry, 1932	1—6 fl., 1—3 fl., 1—5 fl., 6—2 fl., 4—1 fl.	Yes	Yes	3 bldg. old	New unit $3,250,000
22	3	1931		1—4 fl., 2—2 fl.	Yes	No	No	
23								
24	3	1912, 1918, 1916		1—3 fl., 1—2 fl., 1—? fl.	No		Yes	$58,000
25								
26	1	1907		1	Partly	No	No	$132,000
27	1	1885		2	No	No	No	$3,250
28	1 and garage			2 and attic		No	No	$10,000 with land
29	1	1906	1926	2	Yes			
30	1	1929		1	Yes	No	No	
31	2	1916, '26		3	Yes		Yes	
32	1	1928		2	Yes			$270,000
33	3	2—1889, 1—1919		2	No	No	Yes	$60,000
34	19	Attached			Yes	1 bldg.	Yes	Attached
35	7	1925, 1933		2—6 fl., 1—3 fl., 3—2 fl. Power plant	Yes	Yes	Yes	$5,000,000
36								
37								
38					Yes	Yes	Yes	$1,523,050
39	2	1901, '27		2 and 3	Yes	No	Yes	$115,000
40	9	1902	Nurses' homes 1924	1—3 fl.	No	No	Yes	?
41	27	25—1909, 2—1922		12—1 fl., 10—2 fl., 5—3 fl.	Yes		Yes	$3,000,000
42	1	1918		1	Yes	No	No	
43	2	Rebuilt 1935		1	Yes	No	Yes	$100,080
44	10	6—1910, 1—1912, 3—1930	1932	3—3 fl., 2—1 fl., 1—4 fl., 4—2 fl.	Yes		Yes	

TABLE V—CONT'D

SUPERINTENDENT (B. BED CAPACITY)

HOSPITAL	TOTAL NUMBER BEDS	CRIBS (UNDER 2 YR.)	BEDS 2-14YR.	TOTAL NO. IN WARDS	HOW MANY IN CUBICLES	SPECIFY IF NOT ALL	BEDS IN EACH CUBICLE	ANY TYPES CONTAGIOUS CASES EXCLUDED	SPECIFY
1	400	None	Cots to 6 yr. then hosp. beds	54			1	No	
2									
3	310		310	8	All	Exc. 30 rms. of 2-4 beds. Iso. and pri. rooms	1	Yes	Smallpox
4	155	35	120	16-18 pts.	All ex. 1			Yes	Smallpox
5	100	20	80	30	2	Originally sep. for diph.	2	No	Accept measles, chickenpox, mumps, etc., wh. complications or transferred
6	145	90	55	4	2	Observ.	1 or 2		Ven. dis. only
7	117 36 for T.B.					Grd. fl. 9 each; 2 and 3 fl. 20 each	1		Ven. dis. only
8	311	42	50	3	1		1	Yes	Diph. and smallpox
9	30		14	4	4		4	Yes	Smallpox
10	50	10		None					Pertussis
11	65	25	40	3	None	1 to be		Yes	Impetigo, scabies, gonorr., and syph.
12	20	1-6	14-20	3	None			No	
13	74	Interchangeable		13	None			Yes	Typh., pneumo., ven. dis., tbc.
14	38	Interchangeable		2	All		1 and 2	No	
15	45	Interchangeable		3	None			Yes	Diphtheria
16	40	10	30	4	None			Depending on Bd. Hlth.	
17	6-12			1					
18	100	50	50	4	4		1	No	
19	150	Interchangeable							Ven. dis.

TABLE V—CONT'D

20		18	50	4	None			Yes	Diphtheria, sc. fever without reservation
21	550	To 7 yr. 120, 158 in new unit	7-14 yr. 66	22 wds. on ea. floor	Wds. in old bldg.	New unit in rooms	1 and 2	No	
22	176	100	76	14	None			No	
23								No	
24	110	35	75	2	All		4	Yes	Syphilis and typhoid fever
25									
26	60	4	56	2	None	Small rms. with 2 beds		No	
27	14	6	2 jr. 6 adult	2	None			No	
28	18	6	12	2	None			No	
29	35	8	27	3	1	Infants	1	No	
30	16	10	6		All rms.		1 in 12 2 in 2	Yes	Erysipelas
31			50	4	3	1 wd. in single iso. rooms	1	Yes	Smallpox (always), chickenpox and meas. (at times)
32	84	42	42	4				No	
33	38	6	32	4	2	Mixed inf. bldg.	1	No	
34	424			23	17½	12 in Pav. IV; 52 in II	1	Yes	Gonococcus inf., syphilis, erysipelas
35	519			21	18	Pri. floors	4	No	
36									
37									
38	550		To 12 yr. 36	59	2		Av. 4	No	
39	80	8	72	8	None			Yes	Confined to sc. fever and diph.
40	150	25	125	4	None			Yes	Certain types due to lack of isolation facilities
41	907	600	307	22	16	Ex. 6 cottages w. 10 pts. each	2-8	No	
42	12	3	9		12		1	No	
43	50	10	40	4	None			No	
44	265	65	100	9	2	2 wds., single rms.		No	

TABLE VI

SUPERINTENDENT (C. WARD CAPACITY DETAILS)

HOSPITAL	OBSERVATION					CONTAGIOUS DISEASE WARDS							
	SITUATED	NUMBER SEPARATE ROOMS	NUMBER BEDS IN EACH	DIVIDED IN CUBICLES	NO. BEDS IN EACH	NO. WARDS	BEDS IN EACH WARD	TOTAL NO. BEDS	NUMBER CUBICLES	NUMBER SEPARATE ROOMS	SUNLIGHT	OUTDOOR PORCHES	ADULTS AND CHILDREN IN SAME WARD
1	Easy access of adm. room				1	Foregoing information relates to contagious disease					Yes	Yes	No
2													
3	Ends of ea. fl.	60	16 sgl. 32 dbl. 12 4bd.	Yes	1						Yes	No	No
4		12	2	Yes	1 and 2	7	16	127	10		Yes	No	Yes
5	Grd. fl. ambulance entr.	8	1			5	8	40	4	8	Wards have sun gallery	No	No
6	No. and east wings	22	1 cot 1 bed	2	1 and 2	6	14	145	12	22	Yes	Yes	Females and children
7												No	Yes, sep. cub.
8	Cont. Pavl.	4	2 in 2 2 in 1			4	6	24		26	Plenty	Yes	No
9						4	4	30	4	Convertible	Yes	No	No
10		5	1	No		2	20	50			Yes	Yes	Yes
11						3	21, 22, 22	65	None	10	Yes	Yes	Yes
12		2	1-3	No		2	8	20	None	2	Yes	Yes	Yes
13						13	2-6	70	None	36	Yes	Visiting only	Adults and child. not same
14						2	12 rms. 2 bds. ea.	38		14	Yes	Yes	No
15						3		45	None	14	Yes	Yes	Yes
16	Sep. rms.	7	1	No		4	8	32	None	7	Yes	Yes	Yes
17													Yes
18						2	16	32	16	12	Yes	No	Yes
19													

TABLE VI—CONT'D

20													
21													No
22	1st and 2nd floors		3	None		10	14 & cribs	164	None	8	Yes	No	Yes
23		5	1 and 2	None		4			None	8	Yes	Yes	Rarely
24										14	Porches	Yes	No
25													
26		10	2	None		2	8	16	None	10	Yes	No	No
27						2	4	14		3 rms. may be used for 2 bd. wds. or pri.	Yes	Yes	No
28						2	5	10	None	3 semipri.	Yes	Yes	Yes
29						3	4 in 2 8 in 1		1 wd. with 8 cu.	19	Yes	Yes	No
30	All cub.	14	1 in 12 2 in 2	Yes		All cub.					Yes	No	No wds.
31	1st fl.	10	2			3	12	36	12			Yes	Yes
32						4	21	84		4 pri. on ea. wd.	Yes	Roof & yd. on occa.	Yes
33	2nd fl.	6	1	No		4	2-4 & 5	32	9	6	Yes	2	No
34	Pav. III, 1st Pav. IV	72	1	No		23	16, 10, 20	352	297	None	Yes	Yes	Yes, if no sep. room available for type of dis.
35						2	30 & 17	47	6	23	Occasionally	No	No
36													
37													
38		20	1	No		4		30	No	7	Yes	No	Yes
39	1st fl.	1	6	No		8	8	64		9	Yes	Yes	No
40						4	Av. 35	150	None	6	Yes	No	Yes
41	1 east end 2 west end	35	6 in 27 8 in 4 9 in 4			13	44 in 1 45 in 4 50 in 8	624	17	3	Sun parlors		Yes
42						None							
43						4	7-9		None	7	Yes	Yes	Sometimes
44						6	18-35	150		18	Yes	2 wards	Yes at times

TABLE VII

SUPERINTENDENT (D. ATTENDANCE)

HOSPITAL	PATIENTS ADMITTED			PATIENTS DISCHARGED			DIED IN HOSPITAL			NO. OF PATIENTS OPENING OF LAST YEAR	NO. OF PATIENTS CLOSE OF LAST YEAR	COLORED AND WHITE ACCEPTED	IF SO, SEGREGATED	LIMIT TO NO. COLORED CHILDREN AT ONE TIME
	1932	1933	1934	1932	1933	1934	1932	1933	1934					
1		971	963		910	938		29	30	103	105	Yes	No	Very few
2														
3			2,571			2,196			100	Hosp. opened 1/5/34	275	Yes	No	No
4		1,075	1,559		1,064	1,487			35	126	158	Yes	No	No
5			346			316			23	33	40	Yes	Yes	No
6	758	644	575	762	627	539	27	27	23	23	36	Yes	No	No
7	524	438	699	555	402	736				68	31	Yes	No	No
8			5,687			5,646			236	161	202	Yes	Yes	No
9	222	229	298	201	209	265	15	16	30	6	6	Yes	Yes	No
10	156	94	79	151	86	73	5	8	6	3	3	Yes	No	No
11	559	522	491	475	470	472	49	31	27	21	35	Yes	No	No
12												Yes	No	
13	1,453	1,382	1,423	1,369	1,311	1,312	84	71	111	65	65	Yes	Yes	Limited by amount of bed space
14	89	117	167	89	117	152	2	3	8	8	15	Yes	No	No
15	165	101	87	144	73	64	1	3	1	24	23	Yes	No	No
16	135	247	420				7	14	8	15	29	Yes	No	No
17	No answer													

TABLE VII—CONT'D

18	291	261	226	260	258	206	24	18	10	6	17	Yes	No	No
19	972	704	598	944	684	575	28	20	23	49	57	Yes	No	No
20	284	112	100	284	112	100	2	1				Yes		
21	2,701	3,296	2,272	2,559	3,198	2,175	105	122	84	137	147	Yes	No	No
22	729	675	646	799	652	639	12	10	9	59	48	Yes	No	No
23												Yes	If possible	No
24	738	493	402				23	19	18	42	20	Yes	Yes	No
25	91	57	68	94	60	66						Yes	Yes	No
26	Same as 1934		45	Same as 1934		45			None	2	5	Yes	No	No
27	57	280	99	46	276	101	2	3	3	8	3	Yes	Yes	No
28	65	118	63	60	117	64	2	4	1	6	4	Yes		
29	303	374	328	297	374	318			6	11	15	Yes	No	No
30	233	214	164	218	203	155	14	15	5	10	14	Yes	No	No
31	785	669	559	730	624	530	41	43	35	43	37	Yes	Yes	No
32	533	428	269	511	428	267	24	19	12	25	15	Yes	No	No
33	129	156	132	124	143	136	9	10	7	13	2	Yes	Yes	No
34	5,946	6,448	4,689	5,718	6,270	4,429	193	231	195	249	314	Yes	No	No
35		Strong and Roch. 11,357	Strong and Roch. 11,892					Strong and Roch. 556	Strong and Roch. 552	Strong and Roch. 362	Strong and Roch. 375	Yes	No	No
36														
37														
38	9,192	9,456	10,174	9,165	9,399	10,155	361	409	428	344	419	Yes	No	No
39	235	410	251	231	405	250	4	5	1	22	7	Yes	No	No
40	1,108	1,195	1,078	1,019	1,166	1,004	67	54	70	70	74	Yes	Yes	No
41	5,406	3,385	3,286	5,716	3.719	3,425	120	66	122	261	247	Yes	No	No
42	100	53	47	81	46	42	19	7	5	1	1	Yes	Yes	No
43	66	60	69	55	48	60	11	12	9	6	2	Yes	No	No
44	2,549	2,321	1,901	2,556	2,310	1,919	129	148	118			Yes	No	No
Totals	36,624	49,602	54,703	35,213	36,721	40,857	1,510	1,374	2,350					

TABLE VII—CONT'D

SUPERINTENDENT (D. ATTENDANCE)

HOSPITAL	GENERAL PROPORTION COLORED CHILDREN	NO. OF STRICTLY CHARITY BEDS	NO. OF STRICTLY PAY BEDS	NO. OF PRIVATE ROOMS FOR CHILDREN	NO. OF PRIVATE ROOMS FOR ADULTS	FACILITIES FOR MOTHERS TO STAY IN HOSPITAL WITH CHILDREN
1	4 in 1,000	380-385	to 15	Same	Same	No
2						
3	None in 1934	290	20			Yes
4	1 in a yr.	155	?	9	20	Yes
5	1 or 2 a yr.	0	All	6	1	1 suite suitable
6	Very few	120	20	10	10	When nursing
7	Not important enough	According to ability to pay or not				Yes
8	40%	About 30 used by community chest or part pay	239	Used as found necessary	110	Yes
9	10%	No distinction		6	Same as for children	
10	Very few	Municipally owned		5	Same	No
11	1-2%	None		10	Same	No
12		As occasion demands		No	No	No
13	1 to 7 or 8	Available to citizens without pay		According to demands		No
14			14	14		No
15	Rarely have one	45	0			No
16	Rarely 1 in 10 yr.	Interchangeable		7	7	No
17						
18				12 for adults and children		No
19	Few in a yr.	71% unpaid 1934	29% paid in 1934	Not operated for private patients		No
20	Very few	All				
21	1 to 5 white					No
22	2%	All	None	None	None	No
23						No

TABLE VII—CONT'D

SUPERINTENDENT (D. ATTENDANCE)

24	7.7%	All free to Camden res.	All outside Camden pay	None	None	No
25		25	5			No
26	⅓	90%	5%	Not limited		No
27	None admitted yet	None	None	3 for children or adults		No
28						Yes
29	Very few			Rooms for children and adults		Yes
30	1%	All	None	None	None	No
31	3%	All	None	None	None	No
32	1%	Maintained by city	As required			When children critically ill
33	0.1%	36	2	2	2	No
34	10.8%	Municipal Hosp. class not considered	None	None	None	No
35		Divided according to condition	If able to pay expected to do so	3	3	Noncontagious condition only
36						
37						
38		304	246	7	73	Not in contagious ward
39	2%	All pay bills if patients are able, otherwise free		9	9	Only in emergency
40	5%	150	None	None	None	No
41	10%	All	3	3 for both children and adults		No
42	4 in year	Collect whenever possible		12 when needed	9	Yes
43	Less than 5%	Based on ability to pay		7 for children and adults		Yes
44	2%	90%	10%	25	30	Not in wards

admitted 9,192, 9,456, and 10,174 in the three years. Four hospitals admitted about 100 cases annually. In nine hospitals the admissions were 1,000 or over.

The percentages of deaths for all hospitals were 4.1, 2.6, and 4.3 for the years 1932, 1933, and 1934, respectively.

Of 40 hospitals replying, all accept colored patients. In 12 cases the races are segregated. In no case is the number of colored patients limited. The percentage of colored inmates varies from 0.1 per cent to 40 per cent. In 24 of the 40 hospitals the colored patients were under 10 per cent of the total number of inmates.

In answer to the question of availability of charity beds, 33 hospitals replied, of which 5 replied that all their beds are charity beds. Five are city hospitals with free beds for citizens. Others apparently collect whenever possible. Two have 90 per cent charity beds. (In 6 instances the answer to the question of the number of strictly pay beds was "None.") In 1 instance all the beds are "pay beds."

Thirty hospitals replied to the question of private beds. Twenty-one have private rooms for children or for adults. With the exception of 2 hospitals none permit the mother to stay with her child unless the child is critically ill.

Finances.—Forty institutions answered the question concerning finance: of these 4 derived funds from the county only; 1 from state, county, and city; the remainder from the city only. Seven apparently derive only part of their income from this source. None are entirely endowed. Two receive support from the Community Chest.

The cost of running the hospital for a year naturally varies with its size, the lowest reported (19 replies) being $6,000, the highest being $391,144. The cost per patient per day, exclusive of interest on investment, varies from $1.32 to $6.53. The figures for food, nursing, and supplies per day varied so greatly as to indicate misunderstanding the question. In 24 hospitals the superintendent was a man; in 15, a woman. Nineteen of the men and 2 of the women were physicians. Thirteen of the women were trained nurses.

Nursing.—The number answering this was 41. Seventeen are affiliated with other hospitals as far as nursing is concerned. The number of nurses employed varies from 3 to 250 yearly. In some of the institutions the nurses are drawn from other hospitals, and their time of service varies, usually about three months. In 6 hospitals the term of service is three years. In 23 hospitals only graduate nurses are employed to care for the patients.

Thirty-one institutions require immunization of nurses against diphtheria; 19, against smallpox; 10, against typhoid. Out of 33 replies 4 stated that 4 nurses acquired diphtheria in 1934 and 46 contracted scarlet fever. In 10 hospitals other diseases were contracted by nurses (mumps, measles, chickenpox, and whooping cough).

Training.—The number of day nurses varies from 3 to 161, the night nurses from 1 to 32. The question as to their length of assignment to duty was not answered satisfactorily and will be omitted.

Operating Room.—Thirty-nine hospitals answered this section. Of these, 31 have operating rooms, 1 for emergency only. Seven have none. Five have student nurses definitely assigned to the operating room; they are assigned for periods varying from a week to a month. The number of operations varied, one of the largest institutions recording ninety-eight major operations, 62 citrate transfusions, and 46 direct transfusions. In some of the small institutions only one or two operations were performed.

Diet Kitchen.—Ten hospitals definitely stated that no student nurses were employed in the diet kitchen; 8 hospitals have from one to four. In four cases the food is prepared in a central kitchen which serves the whole general hospital.

Supervision.—Of the 33 hospitals replying, 26 have a nurse in charge of each ward. These are all graduates. Only 4 hospitals answered that undergraduates were also employed. In 21 there are graduate nurses in charge of the wards at night, but in 2 hospitals 1 nurse is responsible for more than one ward. Thirty hospitals replied that they have supervising nurses, usually 1 or 2, but in some cases as many as 12, 13, and 22. These nurses are graduates and there are about the same number of night supervisors.

The question of the occurrence of cross-infection was answered by 31 hospitals. Only 1 diphtheria infection was reported. Eleven hospitals reported scarlet fever cross-infection, as follows: 3 had one case each; 4 had two each; 2 had three each; and 1 had four. Eleven institutions had measles cross-infection; 2 hospitals reported one case each; 2 reported two cases; 2 reported four; 1 reported six; 1 reported nine; 1, thirteen; 1, eighteen; and 1, forty cases. Four institutions had pertussis cross-infection, one or two cases each. Ten reported varicella; 4 had one case each; 1 had three; 1 had five; 1 had twenty-one; 1 had twenty-four; and 1 reported "several" cases. Several reported mumps cases.

X-Ray.—Forty hospitals answered this section (Table VIII). Thirteen replied definitely that they had no x-ray facilities; 21 had their own x-ray plant. The remaining 6 send their patients to other hospitals if x-ray examination becomes necessary. There are full-time men in charge in 13 hospitals. In 19 the department is available at any hour of the day or night; 12 are equipped for high speed radiography with milliamperage as indicated in Table VIII. Twelve are equipped for superficial radiography with voltage as indicated in the table. Six are equipped for deep radiotherapy with voltage as indicated in the table. Fifteen have fluoroscopes; 13 can take stereoscopic plates; 18 have portable machines. In 3 hospitals internes take the radiographs. Only 3 hospitals take radiographs as a routine procedure. Twenty-three replied that x-ray facilities were adequate; 1 replied that they were "fair." The x-ray serves the dispensary in 7 cases and outside physicians in 5. The figures given for the number of plates taken during the year vary greatly and run into such high figures that it is evident that the figures apply to general hospitals whose x-ray facilities are used by the contagious disease hospital. These figures are, therefore, of no value for this investigation.

Laboratories.—Forty-one hospitals answered this section completely or in part. Eighteen have a general director for all laboratories; 8 have none. The remainder have their laboratory work done at other institutions. In only 7 is the director a full-time man.

Pathologic Laboratory.—Thirteen hospitals have full-time directors of the pathologic laboratories. Apparently in many instances pathologic work is done at a general hospital. Twenty hospitals have full-time technicians varying in number from 1 to 7. In 16 hospitals the internes do laboratory work, in most cases just routine. In 1 instance autopsies are done by internes under supervision; in another, special mention is made of blood counting and typing of patients in the wards. Seventeen hospitals consider their pathologic laboratory equipment adequate; 4 do not: 2 need more technicians; 1 says space and equipment are limited; 1 complains of inadequately trained bacteriologists.

Twenty-five hospitals replied concerning deaths and autopsies. In 2 hospitals there were no fatalities in 1934. In 3 smaller institutions there was a total of 14 deaths with no autopsies. However, taking the series as a whole, a surprisingly large percentage of postmortems was obtained. The total number of deaths reported for 1934 was 1,857; the number of autopsies obtained was 1,173, 63 per cent. There are facilities for pathologic research in 11 hospitals.

Clinical Laboratories.—Sixteen hospitals are in the immediate charge of a director, a full-time man in 14 instances. Sixteen have full-time technicians, and in 15 the in-

TABLE VIII

X-RAY DEPARTMENT

HOSPITAL	DIAGNOSTIC FACILITIES	NO. FULL-TIME TECHNICIANS	FACILITIES ANY HOUR, DAY OR NIGHT	EQUIPPED FOR HIGH SPEED RADIOTHERAPY	SUPERFICIAL RADIOTHERAPY VOLTAGE	DEEP RADIOTHERAPY VOLTAGE	FLUOROSCOPY	TAKE STEREOSCOPIC PLATES?	PORTABLE X-RAY MACHINE	INTERNES DO X-RAY WORK	ROUTINE	SPECIAL
1	Gen. Hosp.											
2												
3	Yes	1	Yes	Yes	220	200,000	Yes	Yes	No	No		
4	Yes	Part time 1	Yes	No	No	No	No	No	Yes	No		
5	Yes	2	Yes	No	Yes 90-100 kv.	Yes 200 kv.	Yes	Yes	Yes	No		
6	Yes	2	24 hr.	No	No	No	No	No	Yes	Yes	Yes	No
7	Yes		Yes		Yes		Yes	Yes	Yes	No		
8	Yes	4	Yes	Yes 20 mlm.	Yes, high & low	Yes, high & low	Yes	Yes	Yes	Yes	Yes	
9	Yes	1	Yes	No	No	No	No	No	Yes	No		
10	No x-ray											
11	Yes	2	Yes	Yes 200 mlm.	Yes 85,000	No	Yes	Yes	Yes at Gen. Hosp.	No		
12	No x-ray											
13	Yes	0	Yes	No	No	No	Yes	No	No	Yes	Yes	No
14	Yes	0	Yes	Yes	Yes	No	Yes	No	No	No	No	
15	Sent to outside technician			80 mlm.	100 kv.							
16	Lab. work in Gen. Municipal Hosp.								Yes			
17	No x-ray											
18	Yes	1	Daily 8-4:30 on call at night	Yes 100 mlm.	No	No	No	Yes	No	No		
19	T. B. division has x-ray dept. New building will have its own								Yes			

TABLE VIII—CONT'D

20	No											
21	Yes	1	Yes	Yes 100 ma. 100 kv.	Yes 130 kv. 5 ma.	No	Yes	Yes	Yes	No		
22	X-ray in Hudson County Hospital											
23	Yes	1	Yes	Yes 100 ma.	Yes	Yes 200 kv.	Yes	Yes	No	No		
24	No x-ray											
25	No reply											
26	X-ray done through Atlantic City Hosp. when necessary											
27	No x-ray dept.											
28	No x-ray dept.											
29	Done at Ellis Hosp. or Health Center											
30	Yes		Yes	125 yes	Yes	Not used	Yes	Yes	Yes	No		
31	Yes	No	Yes	Yes	No	No	Yes	Yes	Yes	No		
32	Yes	1 Half time	Yes	Yes 30 ma.	No	No	No	No	Yes	No		
33	No											
34	Yes	Part time 1	Yes	No	Yes 120 kv.	No	Yes	Yes	Yes	No		
35	Yes	3, plus 2% res. staff	Yes	Yes 1,000 ma.		Yes 200 kv.	Yes	Yes	Yes			
36												
37												
38	Yes	2	Yes	Yes 100 ma.	Yes 76-130 kv.	Yes 200,000 kv.	Yes	Yes	Yes	No		
39	No x-ray dept.											
40	No x-ray dept.											
41	Yes	No	Day until 7:00 P.M.	No	No	No	No	No	Yes	No		
42	No	No	No	No	No	No	No	No	Yes on call	No		
43	No x-ray department											
44	Yes	1	Yes	No	Yes 125 k. v. p.	No	Yes	No	Yes	No		

TABLE VIII—CONT'D

HOSPITAL	SPECIFY IF SPECIAL	EQUIPMENT AND FACILITIES ADEQUATE	IF NOT, IN WHAT RESPECTS?	DEPARTMENT SERVE DISPENSARY	OUTSIDE PHYSICIANS	NUMBER OF X-RAY PLATES TAKEN LAST YEAR	NUMBER OF SUPERFICIAL TREATMENTS	NUMBER OF DEEP THERAPY TREATMENTS
1								
2								
3		Yes		No dis.	No	56	0	0
4		Yes		No dis.	No	43	0	
5		Yes		No	Yes			7,712
6		Yes		No dis.	No	33	0	0
7		Yes				Included in total of Vancouver General Hospital		
8		Yes		Yes	Yes	5,500	227	9,686
9		Yes						0
10								
11		Yes		Yes	No	197	0	0
12								
13		Yes		No dis.	No	962	0	0
14		Yes		Yes	Yes		0	0
15	Sent to outside technician							
16		Yes		No dis.	Yes			
17	No x-ray							
18		Yes		Yes	Occasionally	822		
19						2,396		
20								
21		Yes		No	No	2,016	40	0
22	X-ray in Hudson County Hospital							
23		Yes		No dis.		629	2	355
24	No x-ray							
25	No x-ray							
26	X-ray done through Atlantic City Hospital when necessary							

TABLE VIII—CONT'D

27	No x-ray department							
28	No x-ray department							
29	Done at Ellis Hospital or Health Center							
30		Yes		No			0	0
31		Yes		No	No	231	0	0
32		Yes				118	0	0
33		Portable x-ray of outside Roentgenologist available					0	0
34		Yes		Dis. for employees only	No	3,617	1	0
35	Assoc. res. radiol.; assist. res. radiol.	Yes		Yes	No	28,049	Total 3,352	
36								
37								
38		Yes		Yes	Yes	10,000	200	Installed in last 2 mo.
39		Yes		No				
40								
41		Fair		No		200	0	0
42		Yes	Send detailed x-ray studies to disp. Wilkes-Barre Gen. Hosp.	No		0	0	0
43								
44		Yes		Yes	No	3,600	40	0

TABLE IX

GENERAL INFORMATION (1934)

HOSPITAL	DIPHTHERIA		SC. FEVER		MEASLES		WH. COUGH		SMALLPOX		CHICKENPOX		EPIDEMIC CEREBRO-SPINAL MENINGITIS	
	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS
1	18	0	647	9	18	0	91	1	0	0	65	0		
2														
3	213	23	772	24	823	7	391	34			96			
4	93	4	792	11	210	5	313	11			38	1	0	0
5	11	0	64	0	2	0	17	3	0		4	0	3	1
6	33	0	245	5	2	0	29	4			20	0	1	0
7	14	4	599	3	0	0	4	0	0	0	12	0	2	0
8	4	0	382	6	23	0	0	0	0	0	6	0	1	0
9	56		61		47		30						8	
10	6	3	43	1	11	1					3	1	1	0
11	18	0	235	2	3	0	14	2	0		6	0	2	1
12	4	0	165	1	18	0	394	1	0	0	574	0	2	2
13	66	6	552	7	294	43	110	26	0	0	11	0	4	1
14	6		31		33	1	19	2	0		7	1	1	1
15			34	0	6	0	1	0	0		0		0	
16	2		304	4	9		2	1	0		4		0	
17	No answer													
18	3	0	167	1	138	0	368	0	0	0	762	0	6	5
19	23	1	432	9	603	35	36	6	0		6	0	0	
20	0		66		0		0		0		0		0	
21	40	1	596	7	28	0	161	8	0		144	0	11	2

TABLE IX—CONT'D

22	71	2	71	1	3	0	32	1	0	0	40	0	4	1
23	1		181	1	9	1	5		0		1		1	
24	56	7	198	5	33	3	19	2	0		0		1	1
25	26	0	56	0	106	0	95	0	0	0	108	0	2	2
26	0	0	27	0	5	0	0	0	0	0	1	0	In A. C. Hosp.	
27			93	3	7									
28	0		63	1	0		0		0		0		0	
29														
30	14	0	95	4	1	0	16	0	0	0	13	1	1	0
31	64	11	366	3	12	2	60	7	0	0	4	1	2	1
32	5	1	143	4	17		52	1	0		5		5	1
33	0		89	1	3		2	2	0		1		0	
34	579	29	1461	8	372	5	505	24			454	0	13	2
35	7	1	141	7	8	0	36	6	0	0	11	1	1	0
36														
37														
38	31	4	54	0	4	0	19	1	0	0	16	2	0	0
39	12	1	238	0	0		0		0		0		0	
40	203	29	615	11	5	0	0		0		3	0	16	12
41	164	21	2348	22	307	34	137	29	0	0	70	1	0	
42	9	1	8	0	1	0	2	0	0		2	0	1	1
43	8	2	38	2	3	0	5	1	1	1				
44	39	1	196	3	13	0	98	7	0	0	19	0	2	1
Totals	1899	152	12,671	176	3783	103	3063	200	1	1	2506	9	91	35
Deaths %	8.0		1.3		3.1		6.5		100.0		0.35		38.3	

TABLE IX—CONT'D

GENERAL INFORMATION (1934)

HOSPITAL	TYPHOID FEVER		AMEBIC DYSENTERY		BACILLARY DYSENTERY		OTHER CASES		
	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS		CASES	DEATHS
1	4	0	8						
2									
3							Erysipelas	84	6
							Mumps	47	
4							Erysipelas	58	8
5	6						Erysipelas	35	1
							Tuberculosis	43	8
6									
7	8	0					Mumps	5	0
							Enceph. lethargica	2	0
							Erysipelas	24	1
							Ant. poliomyelitis	4	0
							Leprosy	1	0
8			2	0	0	0	Erysipelas	1	1
9							Erysipelas	5	
							Poliomyelitis	8	
10							Scabies	9	0
11	1	0							
12	35	5					Influenza men.	2	2
							Influenza	7	0
							Pulmonary tbc.	38	18
							Others from tbc.	7	6
13					12	2	Erysipelas	31	3
							Acute polio.	8	0
							Mumps	6	0
							Gon. ophthalmia	25	0
							Others	304	23
14					1		Mumps	1	
15							Tuberculosis	41	1

TABLE IX—CONT'D

16									
17									
18	5	0							
19							Misc. dis. and doubtful diag.	11	1
20							Unclassified	34	
21	15	0	1	0					
22					4	0	Erysipelas	60	0
							Scabies	144	0
							Flu-pneumonia and grip	46	0
							Imp. contagioso	100	0
							Tetanus	2	2
23									
24							Other diseases	64	
25	3	1	1						
26							Poliomyelitis	1	0
							Erysipelas	5	0
							Croup	1	0
27							Erysipelas	1	
							Septic sore throat	2	
							Septic antrum	1	
28							Erysipelas	6	0
29									
30							Parotitis	6	0
							Pseudocroup	2	0
							Membr. tonsillitis	5	0
							Poliomyelitis	1	0
31									
32							Erysipelas	4	
							Mumps	15	
33									
34	2	0							
35	3	0	4	0	20	1			
36									
37									

TABLE IX—CONT'D

HOSPITAL	TYPHOID FEVER		AMEBIC DYSENTERY		BACILLARY DYSENTERY		OTHER CASES		
	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS		CASES	DEATHS
38	4	1			6	1			
39							Erysipelas	1	0
							Mumps	1	0
40							Erysipelas	59	11
							Diphth. carrier	70	0
							Infantile paralysis	1	0
							Mumps	2	2
							Cellulitis	1	0
41							G. measles	15	0
							Anthrax	4	0
							Polio.	3	0
							Mumps	28	0
42							Tbc. meningitis	2	2
							Polio.	1	0
							Scabies	2	0
							Bronchopneumonia	1	0
							Erysipelas	10	1
							Strep. thr.	1	0
							Mumps	1	0
							V. angina	1	0
43							Influenza	1	0
							Scabies	2	0
							Tbc.	2	2
							Other diseases	6	1
44	7	0					Other diseases	393	29
Totals	93	7	16	0	43	4			
Deaths %	7.5		0		9.3				

ternes do the laboratory work, in most instances only routine. Nine hospitals have a "fume hood"; 13 have a colorimeter. Nine are equipped to determine basal metabolic rates; 17 consider their facilities adequate; 6 do not. The complaints are lack of facilities in the hospital: inadequate space, insufficient apparatus, and technicians. Eleven have research facilities.

Bacteriologic Laboratory.—Twelve hospitals have full-time directors; 11 have part-time directors. Seventeen have full-time technicians. In 17 the internes do routine work; in 3 they do special work. Eighteen have electric incubators, and 1 has a gas incubator. Fourteen hospitals keep animals for experimental purposes, and 12 are equipped for research work. Five hospitals replied that their facilities were not adequate.

Serologic Laboratory.—Out of 27 hospitals replying, 11 each had a director of the laboratory. In the remainder the work was done at other hospitals or by the board of health. Eleven have full-time technicians. In 6 the internes do laboratory work—only routine. Wassermann tests are done in 10 hospitals; Kahn tests, in 4. In 14 hospitals facilities are considered adequate; in 6 they are not. In 11 there are facilities for research.

Miscellaneous Facilities.—Fourteen hospitals have facilities for taking simple photographs; 18, for color photographs. Fourteen have electrocardiographs, of which 1 is owned by a staff physician.

Diet Kitchen.—Out of 39 replies, 19 report dietitians in charge of the diet kitchen. In some cases the person is simply a cook; in others, a nurse, a housekeeper, or a superintendent takes care of the diets. Of the 19 so-called dietitians, 14 apparently have had adequate training. In 27 hospitals the dietitian has entire charge of the menus. Standard special diets are drawn up by the medical personnel in 14 instances; by cooperation between dietitian and physician, in 6; in 12 instances, by the dietitian alone.

Social Service.—Only 13 hospitals out of 38 answering have social service departments. Eleven of the 13 have full-time workers in charge, of whom 9 have had special social service training. Eight hospitals have more than 1 full-time worker. Respective numbers are reported as 2, 6, 2, 3, 2, 3, 3, 2. In 13 institutions all cases are investigated. In all these the parents are interviewed at the hospital; supplementary home visits are made in the case of 10 hospitals. Home visits are made in all cases in 5 hospitals. In 10 hospitals histories are taken. Twelve hospitals answered "Yes" to the question of filing social service data on the history chart, abstracts being attached to the notes or filed with history in 14 cases.

The ability of the patient to pay for hospital service is investigated by the social service department in 12 institutions. In 1 this is done by the city collector; in another, by a "hospital adjuster."

In 13 hospitals the social service division arranges for convalescent care. Only 9 hospitals have convalescent homes available, 1 of which admits adults only. Convalescent institutions are used in 8 cases. Foster homes are available in 7 hospitals. Of these, 6 are used frequently; 1, "only rarely." Twelve hospitals complain that facilities for children's care are inadequate. One reports ability to give convalescent care only if a fee is available.

Eleven hospitals have routine follow-up service, and 7 follow cases only under special circumstances. In 10 hospitals the social service department assists in getting patients to return for follow-up. Three hospitals have volunteers for indoor work and for outdoor work. Seven social service departments are entirely supported by hospital funds; 1, only partially. Eight departments are supported by funds from the outside. Three hospitals have special social service committees, in 2 of which physicians are members. In 2 hospitals the committee is represented on the board of managers.

General Information (1934).—Table IX reveals the number of cases of the more usual contagious diseases admitted to these hospitals during 1934, together with total deaths and the percentage death rate in all hospitals for each disease. Information was also requested concerning cases of other diseases admitted by the hospitals, but the replies are too diverse and varied to warrant tabulating.

The mortality figures are of considerable interest, especially when compared with those reported in hospitals of Region III (J. PEDIAT. pp. 142, 143, July, 1936).

The lowest rate is found in chickenpox (0.35 per cent) and the next highest in scarlet fever (1.3 per cent). Measles is next (with 6.5 per cent); typhoid fever (7.5 per cent), and then diphtheria (8.0 per cent). In spite of all the propaganda for the eradication of diphtheria, the hospitals in Region I admitted 1,899 cases in 1934. It is of interest also that the largest number treated in any one institution was at the Willard Parker Hospital in New York City, where 579 cases were admitted. One of the most elaborate campaigns to popularize antidiphtheria vaccinations was carried on in New York City; nevertheless there is a considerable diphtheria morbidity with 29 deaths. The measles mortality is quite low as compared with that in the Middle West (3.1 vs. 6.3). The largest number of deaths reported for any one disease was 200 in the case of pertussis. The variability in the mortality percentage of cerebrospinal meningitis is well shown. Though the total mortality rate is 38.3 per cent, some hospitals had a much lower and others a much higher rate; in 1 institution out of 16 cases 12 died; in 1 hospital 5 out of 6 died whereas in another hospital out of 11 cases only 2 patients died. It is also striking that in all of these institutions there was only a single case of smallpox reported.

WILLIAMS McKIM MARRIOTT
1885-1936

Williams McKim Marriott, one of America's most distinguished pediatricians, died November 11 in San Francisco, Calif., of a streptococcic septicemia following an appendiceal abscess. Dr. Marriott was born in Baltimore, March 5, 1885. He attended the University of North Carolina from which he received the degree of B.S. in 1904. His medical training was at Cornell University from which he received his M.D. degree in 1910. In 1936 he was given the honorary degree of LL.D. by the University of Missouri. During his medical course at Cornell he served as an assistant in the department of biochemistry, and in 1910 he became instructor in biochemistry at Washington University School of Medicine, St. Louis. In 1914 be became interested in chemical problems in pediatrics and was appointed an associate of Dr. John Howland at the Harriet Lane Home of the Johns Hopkins Hospital and Medical School. In 1918 he returned to St. Louis as full-time head of the Department of Pediatrics in the School of Medicine of Washington University, and as pediatrician-in-chief of the St. Louis Children's Hospital. He was made dean of the medical school in 1924, holding these positions until July, 1936. Last spring Dr. Marriott decided to withdraw from active pediatric work and on July 1, 1936, he became the dean of the University of California Medical School in San Francisco, and the professor of research medicine.

He was greatly interested in chemistry and made numerous contributions to the clinical science of pediatrics. His administrative duties as dean had become so time consuming in the last few years that he was able to give less and less attention to pediatrics; and this led him to make such an important change this year. His most important work was in the field of infant nutrition. He possessed a rare and unusual faculty of applying the results of basic scientific research to clinical medicine. He was an excellent teacher and had a strong influence in the development of many of our younger leaders in pediatrics. He was a member of, and had been honored by, many medical societies, including the American Pediatric Society and the American Academy of Pediatrics. He served for many years as associate editor of the *American Journal of Diseases of Children*. His textbook, *Infant Nutrition*, has recently gone into a second edition. Dr. Marriott was one of the ablest of present-day pediatricians and his death at the age of fifty-one is a distinct loss to pediatrics as a whole, to the associates and assistants who have come under his influence, and to his many friends.

News and Notes

The following men have been certified by the **American Board of Pediatrics.**

Carl George Ashley, Portland, Ore.
Einor Hugo Christopherson, San Diego, Calif.
Herbert Everett Coe, Seattle, Wash.
Frank H. Douglass, Seattle, Wash.
Benjamin F. Feingold, Los Angeles, Calif.
Rafael Clarence Harris, Santa Ana, Calif.
Newell Linton Moore, Santa Ana, Calif.
Norman K. Nixon, Los Angeles, Calif.
William Anthony Reilly, San Francisco, Calif.
Arthur Edward Varden, San Bernardino, Calif.
Jerald Samuel Backstrand, Salem, Ore.
Mary Knott Bazemore, Merion, Pa.
Albert James Bell, Cincinnati, Ohio
Adrien Bleyer, St. Louis, Mo.
Maxwell Philip Borovsky, Chicago, Ill.
Julian Deigh Boyd, Iowa City, Iowa
William Leslie Bradford, Rochester, N. Y.
William L. Channell, Oakland, Calif.
Hugh Chaplin, New York, N. Y.
Arthur William Chapman, Glens Falls, N. Y.
Esther Bridgman Clark, Palo Alto, Calif.
Jean V. Cooke, St. Louis, Mo.
Clyde V. Croswell, Memphis, Tenn.
Leonard Tomb Davidson, New York, N. Y.
Leon De Vel, Grand Rapids, Mich.
Leroy Morgan Downing, Cedar Rapids, Iowa
Howard L. Eder, Santa Barbara, Calif.
Richard Cannon Eley, Boston, Mass.
Gertrude Felshin, New York, N. Y.
Manning C. Field, Brooklyn, N. Y.
Mark L. Floyd, Iowa City, Iowa
L. Fernald Foster, Bay City, Mich.
Henry E. Gallup, Brookline, Mass.
Henry John Gerstenberger, Cleveland, Ohio
A. Lawrence Gleason, Oakland, Calif.
Isidore H. Goldberger, New York, N. Y.
Harold G. Grayzel, Brooklyn, N. Y.
Ruth Alline Guy, Peiping, China
Paul György, Cleveland, Ohio
Jeannette Harrison, Los Angeles, Calif.
Alexis Frank Hartmann, St. Louis, Mo.
Walter Heymann, Cleveland, Ohio
Urius Himmelstein, New York, N. Y.
Luther Emmett Holt, Jr., Baltimore, Md.
Rieta Campbell Hough, San Diego, Calif.
Thomas D. Jones, Richmond, Va.
C. Loring Joslin, Baltimore, Md.
Katsuji Kato, Chicago, Ill.
Camille Kereszturi, New York, N. Y.
Thomas F. Krauss, Rockford, Ill.
Clifford Lanier Lamar, Birmingham, Ala.
Gilbert J. Levy, Memphis, Tenn.
Percy Edgar Luecke, Dallas, Texas
John D. Lyttle, New York, N. Y.
Howard Harris Mason, New York, N. Y.
William Patton McDowell, Norfolk, Va.
Preston A. McLendon, Washington, D. C.
Lionel N. Merrill, Detroit, Mich.
Marjorie F. Murray, Cooperstown, N. Y.
Harry A. Naumer, Brooklyn, N. Y.
John D. Nourse, Cleveland, Ohio
Gilbert Seymour Osincup, Orlando, Fla.
M. C. Overton, Lubbock, Texas
Paul L. Parrish, Brooklyn, N. Y.
K. C. Reese, Tulsa, Okla.
Irwin Rubell, Chicago, Ill.
Mitchell I. Rubin, Philadelphia, Pa.
Stanley Stewart Saunders, High Point, N. C.
Béla Schick, New York, N. Y.
A. Clement Silverman, Syracuse, N. Y.
David M. Siperstein, Minneapolis, Minn.
Eugene Herbert Smith, Ogden, Utah
Joseph Stokes, Jr., Philadelphia, Pa.
John A. Toomey, Cleveland, Ohio
Alexander Ashley Weech, New York, N. Y.
William Weston, Columbia, S. C.
T. Wistar White, St. Louis, Mo.
William R. Wilson, New Haven, Conn.
Mendel Zeligs, Cincinnati, Ohio

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